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# THE ALTERNATION OF BLOOD SUPPLY AS A CAUSE FOR NORMAL CALCIFICATION OF BONE

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FOLLOWING fractures, immobilization and disuse cause atrophy of bones. Early activity is known to prevent this decalcification, and it apparently hastens the formation of callus and solid union. The purpose of this study, the results of which are here presented, was to determine the physiological reason or reasons for these phenomena. It has developed the theory that calcification is brought about and maintained by the alternating decrease and increase of the blood supply to the bones. This alternation of blood flow is dependent to a great extent upon the contraction and relaxation of muscles. We often read in medical literature the statement that use of a part will heal a fracture because it increases the circulation. We have accepted this statement without question because of its repetition over a long period of time.

An understanding of our present day knowledge of the factors involved is necessary, and to this end a brief statement is made of the anatomy of the blood supply of bones and muscles, the effects of muscular contraction, the phosphorous calcium balance in blood and tissue, and its relation to the hydrogen-ion concentration. Certain theories of bone formation will be discussed and an attempt made to correlate these facts and theories as they relate to bone calcification and decalcification.

Abnormal metabolic states are not considered, as they do not seem to have a specific bearing on the problem involved.

The bones are poor in capillary nets (17) but contain an abundance of fine arterioles. In long bones arteries supply the spongy structures of the extremities by twigs coming from the articular arteries which branch in the periosteum. The medulla is supplied by the nutrient artery, which passes obliquely into the shaft. These arteries enter the shafts of long bones usually protected and surrounded by a heavy covering of muscle. They are derived mostly from muscular branches and are subject to the same changes that affect muscular arteries during muscular contraction. The nutrient artery of the humerus (7), for instance, arises from the brachial artery in the middle of the arm or one of the muscular branches, passes downward, pierces the tendon of the coraco-brachialis, and enters the humerus below the insertion of that muscle.

The main arterial trunks of supply for the extremities, for the most part, are placed in such relationship with surrounding structures that muscular activity will cause as little pressure as possible upon them. The muscular and nutrient arteries of the bone are not protected in a like manner.

The femoral artery in its proximal half lies in the femoral trigone and is comparatively

superficial; then for a distance of 31 millimeters (1¼ inches) it is enclosed in a fascial sheath. It emerges in the popliteal space as the popliteal artery again becomes quite superficial.

The axillary and brachial arteries are superficial and so placed as to be little affected.

Hyperemia causes decalcification of bone (15). If any process, even in the vicinity of bone, increases the activity of the circulation it will cause rarefaction and resorption of calcium. Ischemia, or decrease in circulation, favors calcium deposition and ossification.

As Grieg says—

1. Maintain the circulation within certain limits and bones remain unchanged.

2. Produce a definite hyperemia and bone undergoes rarefaction, decalcification, osteoporosis.

3. Restrict the blood supply and bone undergoes consolidation.

Hyperemia, either traumatic or infectious, brings about decalcification, the common and striking example being the atrophy of the bones of the hand during the course of an infection in this region. Often erroneously diagnosed as osteomyelitis, recalcification takes place as the infection and hyperemia subside and normal activity is resumed.

Syphilitic osteitis with its accompanying bone sclerosis is based on the obliteration of terminal arteries and the consequent ischemia. Acute infectious osteomyelitis is accompanied always by early rarefaction due to the initial hyperemia. Later thrombosis of arteries occurs. The periosteum, due to edema, is lifted from the bone and its circulation interfered with; then recalcification and sclerosis occur as the inflammatory reaction subsides and fibrosis takes place. A fragment of bone (12), such as the femoral head or the astragalus, completely cut off from its blood supply so that it is completely avascular, will retain the original calcium content. It is often said that the intra-articular fractures of the neck of the femur do not heal because of lack of blood supply. It is probably due to traumatic hyperemia caused by movement of the fragments; fixation by the Whitman method is not accurate and the constant turning of the patient and unavoidable movements in the cast produce irritation of the fracture surfaces. As later and more complete methods of internal fixation are being employed, better end-results are being reported. As a proof that hyperemia is the cause of non-union, it is of interest to note that the head of the femur, with an admittedly poor blood supply, is not absorbed—and this too, when from an x-ray standpoint it is alive; while the neck, particularly the distal portion, is completely absorbed in almost every instance. Kolodny reached the conclusion that the ligamentum teres in the aged is of no perceptible importance, but that the periosteal vessels of the neck are of the greatest importance for the nutrition of the head and neck.

Almost all pathological changes in bone roentgenograms are interpreted on the basis of blood supply, inasmuch as a decrease of blood supply produces a more dense, sclerotic bone, while an increase above normal brings about rarefaction and a consequent lessened resistance of the x-rays. Highly malignant neoplasms of bone are always very vascular and at the same time rapidly destroy and decalcify bone. Less malignant sarcomas of osteogenic origin are of fibrosing type with a minimal vascularity, grow slowly, and are at times accompanied by bone deposition instead of destruction. Exposure to therapeutic doses of x-ray produces a fibrosis, a contraction of blood vessels, and recalcification of bone.

When a normal healthy individual receives a broken leg necessitating confinement to bed, a generalized decalcification takes place. In the fractured limb a more pronounced atrophy takes place, especially if the extremity is confined in a plaster cast. This bone absorption can be due to just one thing—the lack of muscular activity. The atrophy in the unconfined, uninjured extremities, even after three months of bed rest, is hardly discernible; in the incased limb it is often marked. The difference can be accounted for, neither by weight-bearing, nor stress and strain, but only by the fact that there is a difference in muscular activity of the parts involved. To estimate the importance of muscular activity, a clear conception of the rôles of muscular contraction and relaxation is necessary, and particularly of their effect on the circulation.

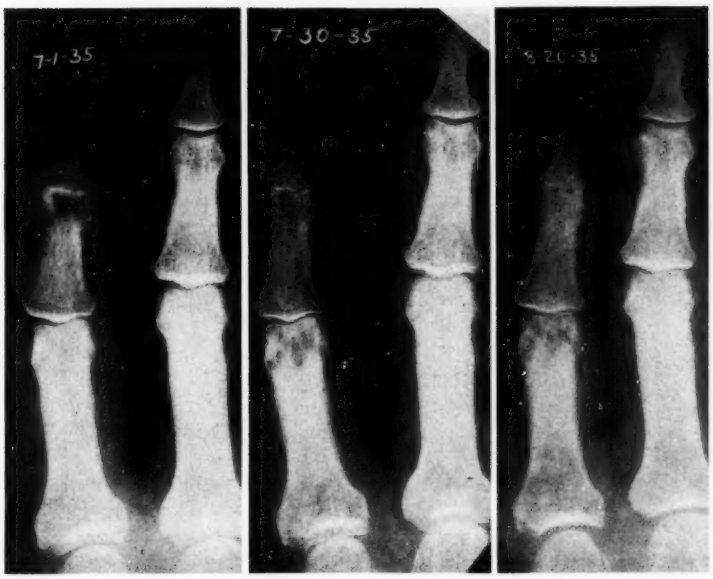


Fig. 1. This patient, a milkman, was referred for operation or amputation on account of an infected index finger of 6 weeks' duration. A diagnosis of osteomyelitis had been made by his attending physician. Treatment consisted of continuous hot packs for at least a month. He arrived at my office carrying a hot water bottle to ensure the continued application of heat. Only a small sinus was present on the dorsum of the index finger near the second interphalangeal joint. The hot packs were discontinued. Three days later the sinus had healed. He was then advised to return to his former occupation which required milking 15 cows twice a day. The alternating contraction and relaxation of his hands in milking produced an alternating ischemia and hyperemia with the remarkable recalcification seen in these dated x-rays.

Contraction of muscle (1) causes a diminution of the flow of arterial blood into the muscle. Relaxation causes an increased supply to enter the muscle. At a certain strength of contraction the arterial system becomes reversed and some blood is at times thrown back against perfusion pressure. Continuing the experiment causes an increase of blood to flow through the muscles.

When a muscle contracts, its form is greatly altered (14). The whole vascular system is beautifully adapted to these changes. The arterial and venous networks insure supply and drainage. The capillaries in resting muscle are straight, in contracted muscle twisted. The blood is driven out of the veins by compression and when muscle relaxes these can refill only from their peripheral ends. The veins are a very effective pump on account of venous valves. The contraction of skeletal muscles (26) squeezes the veins within them and drives the blood toward the heart. Muscles of the extremities are confined in a non-elastic tube composed of fascia and by fascial intermuscular septa to the bone. When muscular contraction takes place the muscles change their form and contour. This causes pressure on the limiting membrane and on the periosteum covering the bones. When we stand, the pressure on the soles of our feet produces an ischemia. When we walk, the activity of our muscles produces alternating ischemia and hyperemia of the lower extremities by the pressure caused by the change of form and contour of the muscles, by the traction on joint capsules and ligaments and periosteum, and by mechanical pressure against muscular arteries and their associated arteries which supply the periosteum and bone. Ischemia of the hand can be seen when the hand is

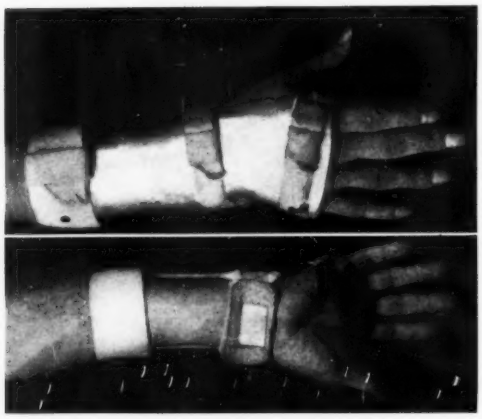

Fig. 2. Posterior aluminum splint used in the treatment of fractures of the scaphoid and other conditions about the wrist joint in which faulty calcification is present. The splint allows alternating contraction and relaxation of the muscles of the hand and forearm. This brings about an alternating ischemia and hyperemia which is so necessary in the production of calcium deposition and callus formation. It is important that the thumb be allowed free movement and that the patient use his hand for all ordinary activities. Shortly after the application of this splint, swelling, heat, and pain—all evidences of traumatic hyperemia—subside and healing takes place in a normal manner.

clenched and a fist is made. When the hand is opened, the blood can be observed reentering the pale areas. The greatest effect of these factors on alternation of blood flow would be on the vascular and compressible periosteum. If alternation is important in normal calcification this might be an explanation of the importance of the periosteum in bone formation. The chemical products of muscular contracture can have no effect on bone metabolism, because they are borne away by the blood stream or destroyed in manufacture of muscle energy.

Y. Henderson and his co-workers have reported the effect that a longitudinal pull of a tonic muscle induces on the transverse pressure between the muscle fibers, and by measuring the force required to inject saline between the muscle fibers have estimated this pressure, both in relaxed, and in one case, in actively contracting muscle. They are of the opinion that the tonus influencing the circulation of the blood is not merely the tonus of the blood vessels, arteries, capillaries or veins, but, in addition, the tonus of the tissue through which these vessels pass. The pressure being maintained, the blood flowing from the arteries into the tissue capillaries does not lose its pressure. The pressure retained by the blood in the capillaries is the force that drives it back into the veins toward the heart —when, on the contrary, in illness or after surgical operations tonus is greatly lessened, the arteriolar pressure may be practically zero in the capillaries and the blood will remain in the tissues. Loss of tonus takes place when a cast is applied. The muscles soften and lose their normal resiliency. A comparative hyperemia ensues. The blood flow is not resisted by the contraction of the muscles, and the venous blood is delayed in its return.

Henderson states that it is a well known fact that during physical exercise any contraction of a muscle squeezes blood into the veins and toward the right heart. They chose a muscle in a state of low tonus for their estimation of intramuscular pressure in man—that of the biceps humeri in relaxation—and noted that if the slightest tension was placed on the muscle, the intramuscular tension was increased markedly.

A hypodermic needle of No. 20 gauge was inserted into the body of the muscle and the force required to inject a small amount of normal saline was measured on a manometer. The statement that tonus produces sufficient pressure in the musculature of the body to be a prime factor in maintaining circulation is original with them, but a closely related idea, namely, that "vigorously contracting muscles produce an internal pressure is as old as William Harvey and Borelli."

They report one experiment performed to determine whether the maximal intramuscular pressure would be sufficient to stop circulation through the muscle. A young man of good muscular development was the subject. His arterial pressures were 120/80 millimeters of mercury. Standing at ease the internal pressure of the gastrocnemius moderately relaxed was 143 millimeters of water. Standing on one toe with the whole weight of the body supported by the leg muscles, the intramuscular pressure rose to 312 millimeters of water; 312 millimeters of water is 24 meters of mercury,

Fig. 3. Six months previous to January 3, 1933, this girl received an injury which caused a fracture of the left scaphoid. Diagnosed as a sprain it was untreated. A posterior aluminum splint was worn for a period of approximately 6 months with the eventual result shown. Fractures of the scaphoid, new and old, will unite if the wrist is fixed but if full movements of the fingers and thumb are permitted.

about ⅓ of the diastolic pressure. This pressure would be insufficient to stop circulation completely.

The general effect of muscular contraction is to retard arterial flow in the muscles and hasten venous return; with relaxation the opposite condition ensues; in other words, alternating relaxation and contraction bring about comparative alternating ischemia and hyperemia.

Diminished blood supply tends to produce an acidosis of tissues; hyperemia a comparative alkalosis. Normal tissue reaction may be considered the same as that of blood; namely, hydrogen-ion concentration 7.3, which, of course, is slightly alkaline.

In a comprehensive series of experiments, Rous (22), using vital stains injected into rats, has shown that skin grafts are acid until their new blood supply is formed. He cites Araki as recognizing that acidity develops in tissues when their blood supply is interfered with. When a string was tied around the legs of a shaven rat stained with phenol red, the color of the leg turned from red to orange in a few minutes, only to become red again a few minutes after the cord was cut.

In another group of experiments (23) rabbits were injected with phenol red and anesthetized. Even under the best conditions, and in the absence of anesthesia, the mere weight of the body of the experimental animal proved sufficient to give rise to acidosis of the surface tissues pressed upon—as shown by the change of color from red to orange—and when the limbs were stretched out under tension the circulation on the surface of the abdomen was frequently interfered with and acidosis developed there as well.

The chemical formula of the mineral portion of bone is supposed to be a double salt of calcium carbonate and tri-calcium phosphate with a formula of $(Ca\ CO_3)\ 2\ Ca_3\ (PO_4)_2$. The blood plasma and tissue fluids (4) hold about the same amount of calcium in both crystalloid and colloidal solution, part of the calcium being ionized and part un-ionized. The condition of the acid-base equilibrium (5) unquestionably is a factor in determining the degree of ionization of serum calcium. The concentration of calcium ions varies directly as the hydrogen-ion concentration. Increase of carbon dioxide tension appears to enable blood to maintain a higher concentration of calcium

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Fig. 4. This boy fractured his left scaphoid 1 year previous to August, 1936. He had been advised to have this bone removed. A posterior aluminum splint was prescribed. Within a week the swelling and stiffness had almost completely subsided. Four months later, union was nearly complete. He has worked using his fingers and his thumb, but at the same time the wrist has been immobilized.

than is ordinarily possible. Howland has shown that a difference of 0.02 in hydrogen-ion concentration of the body fluid is sufficient to determine the site of the precipitation of calcium, and he visualizes calcification in this way: serum contains calcium and inorganic phosphorus in solution in much higher concentration than water would hold them, on account of the high carbon dioxide tension. There is some evidence to show that part of the calcium is protein bound; the calcium is poorly ionized, and this would be equivalent to reducing the total calcium concentration. Intercellular fluid contains roughly the same amount of calcium and phosphorus as the serum, but the protein is much less. The carbon dioxide tension is relatively high on account of cellular activity, but, supposing the carbon dioxide tension is low because the tissue is inactive or dead, conditions are then favorable for precipitation. Cartilage and bone are both inactive tissues and it is in them that one would expect the carbon dioxide tension to be low, and it is in them that precipitation takes place.

Fig. 5. An old fracture of the neck of the femur with non-union and absorption. From an x-ray standpoint the head is alive. There has been little or no absorption of that portion of the neck attached to the head. A spicule on its inferior portion has remained unchanged. The head of the femur has less blood supply than the trochanteric region and the distal portion of the neck. The distal portion of the neck is almost completely absorbed. This is due to a traumatic hyperemia. Increase of blood supply does not bring about union.

Phemister, in a recent article, suggests the possibility of an enzyme carbonase, and calls attention to Hastings' formula for conditions favorable for bone repair. This consists of:

1. Optimum physical conditions for the surface precipitation of calcium salts or surfaces which are provided by newly formed fibrils and cement, and

2. Optimum chemical conditions which are provided by the existence of calcium ions, phosphate ions, and carbonate ions in tissue fluid. In order to have the precipitation of the double salt $(Ca\ CO_3)\ 2\ Ca_3\ (PO_4)_2$ it is necessary that its solubility product constant be exceeded. This might be accomplished by an

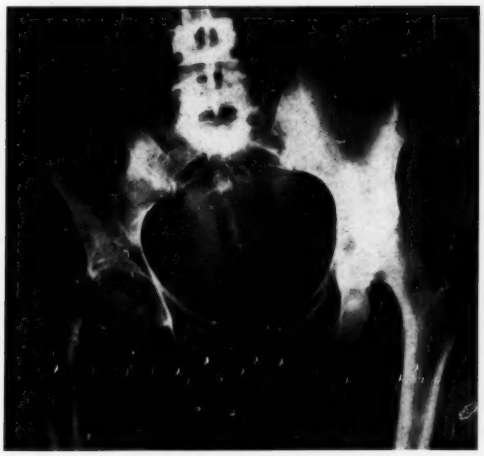

Fig. 6. Fracture of an ankylosed hip treated by the application of a spica cast extending from the axilla to the toes of the affected side. Three months later marked atrophy of both the distal portion of the femur and the head of the bone had taken place. Certainly the head in this case had sufficient blood supply, yet non-union and atrophy occurred in both fragments. The atrophy involved only slightly the normal femur because, although the patient was bedridden, muscular activity and tension were not abolished by fixation.

increase in the calcium, $PO_4$ or $CO_3$ ions. Then by the law concerning difficulty soluble salts the calcium carbonate and the calcium phosphate will be precipitated together.

*Phosphatase.* The discovery of phosphatase by Robison is significant in that it is apparently a very great step forward in our understanding of bone formation and calcification and certain types of abnormal skeletal changes. Phosphatase is an enzyme which has the property of hydrolyzing phosphoric esters present in the blood stream and tissue fluids. He presents the following speculation: A molecule of phosphoric ester is seized upon by the enzyme molecule and combined with it; the alcoholic group is split off, leaving the phosphate group unattached. This then combines with the wandering calcium and carbonate group to form the bone salt. This enzyme is most active in an increasingly alkaline medium, beginning with hydrogen-ion concentration of 7 and reaching an optimum at hydrogen-ion concentration of 8.4. Robison quotes Hofmeister as suggesting that variations in amounts of carbon dioxide in the lymph might account for the precipitation of calcium salts; a higher concentration of carbon dioxide enables lymph to take up more calcium salts.

Fig. 7. Austin Moore's method of internal fixation in intracapsular fractures of the hip. If fixation can be made without the application of a cast this would allow muscular activity of the affected extremity. The fixation should be as near absolute as possible and made with so little foreign material that no reactive hyperemia would ensue. Theoretically under these circumstances, healing should take place in this fracture as readily as in any other.

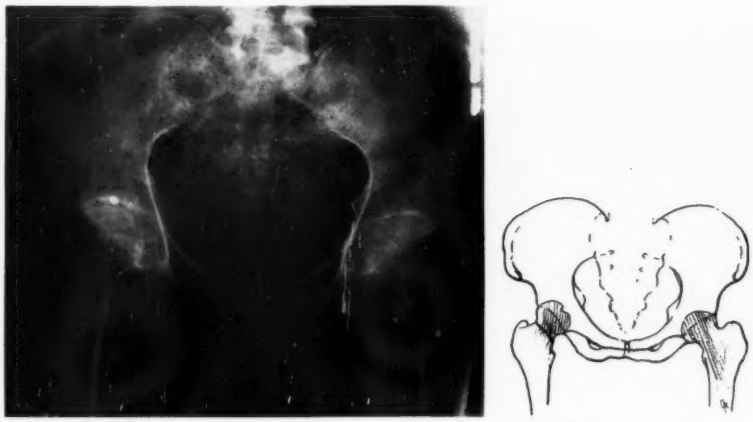

Fig. 8. An impacted fracture of the neck of the femur—the abduction type (Boehler). This fracture healed because the fixation is complete. The blood vessels across the line of fracture are certainly destroyed at the time of fracture but healing takes place without any external fixation because a traumatic hyperemia is not produced by the movement of the fragments upon themselves.

Wiggers states that the theories as to the process of calcium deposits may be grouped as humeral and cellular. According to the former, lime is precipitated as a result of physicochemical changes; according to the latter, the osteoblasts are in some way concerned. The osteoblasts (hypertrophic cartilage cells) secrete phosphatase which hydrolyzes the phosphoric esters and increases the phosphoric ions. (Robison 1926—Kay 1929–1930.)

Wiggers believes that several factors are involved, both general and local—the substrata furnished by the blood, and a local factor. Of the general influences, the content of the blood calcium and phosphate and the hydrogen-ion concentration of the plasma are important. The local ones are a high hydrogen-ion concentration diminishing the solubility of calcium in the presence of phosphate and calcium-ions and the specific effects of phosphatase.

*Circulation.* Normal circulation to bone is a varying circulation, an alternating ischemia and hyperemia. When a fracture occurs, for a while repair is carried on without the benefit of circulatory blood. The hematoma surrounding the fracture (24) becomes acid then alkaline. The acidity of the hematoma leads to the absorption of salts from the bone. The process is at first purely a physicochemical action, then cells grow into the hematoma and redeposit the calcium phosphate in an orderly manner. If the extremity in which the fracture is located is encased in plaster, allowing little muscular function or activity, the part, on account of the lack of muscular tension, really has an increase of blood supply without any alternating ischemia.

Every fracture causes a reactive hyperemia which clinically produces swelling and heat in the region injured. This reaction is brought about by the extracellular presence of a substance called histamine, or the "H" substance of Lewis. This liberated material causes immediate dilatation of the smaller vessels. The reaction is on a more or less physiological basis. Dale has shown that another substance, acetylcholine, is a powerful dilator the action of which is pronounced and long. He says: "We first have the liberation of histamine from the immediately affected cells directly causing vasodilatation of the minute vessels with which it comes in contact and acting as a persistent stimulus to the sensory ending of the terminal axon branching. We must further imagine that the nerve impulses thus engendered, passing through the bifurcation to the ending of the arteriole, there liberate as the effective dilator, acetylcholine."

Fig. 9. Syphilitic bone change February 11, 1925. The increased sclerosis, the result of obliterating arteritis, is the common finding in this disease. The decrease of blood supply causes an abnormal deposition of the bone salt.

In any fracture in which immobilization is not complete two factors are at work: (1) a traumatic hyperemia is set up which brings about bone absorption, and (2) muscular activity producing alternation of circulation and a consequent tendency toward callus formation. Thus union often takes place in spite of movement of the fracture fragments. Union does not take place in cervical fractures of the hip or in fractures of the carpal scaphoid when immobilization is incomplete, because of the small amount of organic material present, which, when irritated, develops a traumatic hyperemia, a dilatation of small vessels such as we see in granulation tissue—the deleterious effect outbalancing the effect of activity.

### EVALUATION

Disuse is known to produce rarefaction and bone atrophy. This rarefaction is due to the absorption of the mineral elements of bone. The cellular constituents remain and as activity is resumed the complicated bone salt is replaced. Activity is due to one thing only—the contraction and relaxation of muscles controlling the extremity involved. It is not due to the chemical by-products of muscular contraction, as they are either used up in the muscular metabolism or swept away by the veins toward the heart.

Rous has shown that ischemia, even slight, such as the weight of the body of the experimental animal, will produce a localized acidosis in the pressure area; as an example he cites the acidosis occurring in the abdominal wall by the slight tension produced by stretching the limbs of the animal. He also notes the speed at which these changes are discernible in the stained animals, only a few minutes being required for the eye to differentiate them.

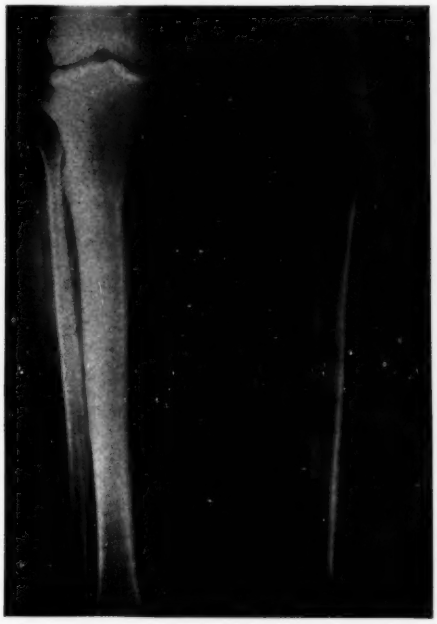

Fig. 10. This patient, a young man now 27 years of age, when an infant, suffered an attack of anterior poliomyelitis, which involved one extremity, completely, below the knee. He walks without support although the affected extremity is somewhat shorter than the normal one. The bones of the paralyzed leg are atrophic and decalcified. The obvious cause is the lack of muscular activity. Involvement of nutritional nerves has been given as the reason for this bony abnormality, but so far as can be determined no such nerve has ever been isolated. The lack of muscular pressure, alternate contraction and relaxation is suggested as the cause of both the atrophy and shortening.

The impression given, although the author refrains from drawing positive conclusions, is that the change toward the acid reaction and toward the alkaline side is accomplished quickly and by very little change in the blood supply.

By the contraction and relaxation of muscles it would seem that these changes could be brought about in bone. It does not seem pos-

Fig. 11. Fasciæ of the thigh. Muscle is surrounded by comparatively inelastic fascia. When muscle contracts the pressure waves create a force at a right angle to the long axis of the muscle while the contraction that ensues creates traction in a parallel direction. These forces cause a mechanical pressure against the periosteum and the vessels supplying the bone.

sible that reossification of bone is produced in any other way; weight bearing and stress and strain can be ruled out because even the slight activity of a person confined in bed will prevent the atrophy of the bone or bones of a limb not encased in plaster.

The hydrogen-ion concentration of blood is known to vary little during health on account of buffer substances which hold the reaction close to 7.35, any great variation being incompatible with life. It is conceivable, however, that tissues and tissue fluid reactions vary to a greater extent during ordinary metabolic activity. Alternating ischemia and hyperemia, therefore, can produce a comparative alternation in the hydrogen-ion content of tissue fluids. It is assumed that during a rest period when muscles are relaxed, particularly immediately following exercise, when arterial flow is markedly increased, a swing toward the alkali side is taking place. Phosphatase acts best in hydrolysis of the phosphoric ester in an alkaline medium. At this period inorganic phosphates should increase in the tissue fluids. Muscular contraction taking place produces an ischemia, a lower hydrogen-ion concentration, and as a more acid reaction develops, more calcium is ionized and the tissue fluids are able to hold in solution a higher supersaturation of calcium. As muscular relaxation takes place and the swing is again toward a higher hydrogen-ion concentration, the law governing difficulty soluble salts causes a precipitation of the complex bone salt, composed of calcium carbonate and calcium phosphate.

### CLINICAL APPLICATIONS

The theory here presented is applicable in a practical clinical way. If hyperemia produces resorption of bone, the use of heat in recent fractures is a wrong practice. Heat produces hyperemia. Skin temperature is generally accepted as an indicator of blood flow to a part.

Heat should be used only when it is the purpose to absorb calcium, as, for example, in calcification of the subdeltoid bursa or other heterogeneous calcifications. The use of heat in acute bone atrophy is a vicious practice, because it increases the pain and absorption bone.

Massage has been praised from time immemorial for increasing the circulation, and certainly after massage the skin is warm and the part to which this treatment has been applied gives every indication of an increased blood flow. It would seem that massage is not indicated before complete union has taken place and all bone atrophy has disappeared. The active use of a limb by alternating the blood supply will help heal the fracture and recalcify the bone. In fractures about the ankle, if accurate reduction is obtained and complete fixation is possible, walking should be permitted since it allows contraction of the muscles of the leg and foot, though the ankle joint does not move. A non-padded walking cast as described by Boehler allows walking without displacement of the fracture.

Contrast baths have been used for years to hasten healing of fractures and to mobilize stiff joints and decrease pain in injured extremities —this usually with the idea that it increases circulation. It is possible this procedure causes an alternation of the blood supply to the part.

Theoretically it would seem that the use of alternating suction and pressure (19), by increasing and diminishing the blood supply, might aid in the production of union.

Rollier has allowed muscular activity in the course of sunlight treatment, believing that it aids in the recalcifying effects of the sun.

Application of this theory to treatment of fractures of the neck of the femur deserves special consideration. The clinical trend is to treat this fracture by internal fixation, by the Smith-Petersen nail, by wires or bone transplantation. In such a fracture of the aged, the blood supply is less than that found at an earlier period in the patient's life. If absolute fixation could be obtained with material which did not cause an inflammatory reaction and absorption of bone, and at the same time use of the extremity and active muscular contraction and relaxation be maintained, the ideal requirements would be achieved. Complete and absolute fixation is necessary in fractures of the neck of the femur—not primarily because the blood supply is so poor that the fracture will not unite, but because the organic elements are few and the slightest movement destroys the organization of the callus. The ischemia present will actually help the precipitation of the bone salt. Also fracture of the neck of the femur occurs in the aged where muscles are atrophic and inactive—so much more the reason for evolving a treatment by which their activity would not be restricted.

Fracture of the carpal scaphoid belongs in some respects to the same category. Fixation is poor by any method. The slightest movement of the hand causes disturbance of alinement with a resultant reactive hyperemia, producing, if continued, cystic degeneration and nonunion. However, if fixation in a dorsal splint is maintained and use of the hand continued, healing will occur in almost every case, even after a long period since the original injury has elapsed.

## SUMMARY

1. A theory that alternating ischemia and hyperemia maintain normal calcification of bone and aid in the healing of fractures is presented.

2. This alternation is produced by the contraction and relaxation of muscles.

3. Certain facts and theories have been cited to support this thesis.

4. Correlation of these facts and theories has been attempted.

5. Clinical application of this theory has been suggested.

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# SALIVARY GLAND TUMORS

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SALIVARY gland tumors are not of rare occurrence. During the 10 year period between January 1, 1927, and January 1, 1937, 81 patients with tumors of this type have been operated upon at The Lahey Clinic. McFarland had collected 301 similar cases up to 1936 and other large series have been reported (1, 3, 9, 11). In the published articles on tumors of this type, however, considerable confusion still exists concerning the histogenesis, pathology, treatment, and prognosis of this disease. In an attempt to obtain further knowledge of this type of tumor, we have reviewed and are reporting this group of 81 cases.

The cases have been grouped for analysis according to the pathological condition present. We have obtained follow-up data on all but 6 of the patients. In this paper we wish to discuss the histology and histogenesis of tumors of the salivary gland as has been brought out by this study and to discuss from a clinical point of view the treatment which we believe should be employed in patients with tumors of this type, and the results which may be expected.

As will be noted from the individual case reports at the end of this discussion, 72 of these tumors are classified as benign and 9 as malignant. The majority of the benign tumors are the so called "mixed tumors," of which there were 51 in this series (Figs. 1 to 5).

Few tumors present more variable and complicated histology than those of salivary gland origin, and there is still much discussion as to their histogenesis. The present inclination is to consider them as adenomas with varying types of stroma, the characteristic appearance being brought about chiefly by the discharge of secretion of a mucoid nature from the epithelial cells into the interstices of the stroma.

Attention has been called to the similarity of the mucoid changes in the stroma of the so called mixed tumors to the mucoid changes in the stroma of basal cell carcinomas. On careful study, however, a distinct difference is found in the character of this change. In the case of the so called mixed tumor the stroma is often distinctly myxomatous, whereas in the case of the basal cell carcinoma it is usually a mucoid degeneration of pre-existing collagen.

The frequent tendency of epithelial cells to take on a spindle form has also been used to explain the apparent mesenchymal structure of certain of the elements of the so called mixed tumors. There are, however, certain objections to the consideration of the mixed tumors as adenomas with peculiar stromal changes. Adenomas readily recognized as such are extremely rare in the salivary gland. Moreover, this particular type of change in the stroma is almost unknown in adenomas developing elsewhere. It seems peculiar that in this group of glands alone a peculiar stromal change should develop in the adenomas which arise from them. In the very few instances of the true adenomas which have been reported there is no suggestion of the structure seen in the mixed tumor.

The concept of a true mixed tumor developing from embryonic tissue is far more appealing. Distinctly in favor of this is the not infrequent occurrence of mixed tumors in diverse localities, such as the hard palate, the tonsillar fossa, the soft palate, the cheek, or the gum. Why adenomas of the salivary gland should develop in these localities and then undergo a peculiar change of their stroma in addition, is a little difficult to understand. Moreover, in those instances in which mucinous material is secreted by epithelial cells and discharged into the stroma, best seen in the mucinous or colloid carcinoma, the picture does not resemble that of the mixed tumor of the parotid gland. Instead of a diffuse change in the stroma, masses of mucinous material spread apart the stromal bands and collagenous material which maintains otherwise its normal characteristics.

The demonstration in parotid tumors of mucinous secretion and its discharge into the

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Fig. 1.

Fig. 2.

Fig. 3.

Figs. 1, 2, 3, 4, and 5. Mixed tumors of the parotid gland.

Fig. 4.

Fig. 5.

surrounding tissue, as emphasized by Fry and Zymbal, has no significance in explaining the histological picture other than emphasizing the reason for the clear spaces surrounding the epithelial cells, which have frequently been mistaken for the lacunæ of cartilage cells.

The hypothesis that the mixed tumor of the salivary gland arises from embryonically misplaced tissue was developed by Cuneo and Veau, who felt that the complex embryonic development of the face gave ample justification for this type of origin.

Many of the tumors can quite reasonably be derived from branchial clefts. If this origin of the tumors is assumed, their extraordinary variability of histological appearance is well accounted for. This view was ably expounded by McFarland.

In this group of tumors at present under discussion, we feel the evidence to be definitely in favor of embryologic misplacement of cells as the mode of origin. In this particular group we have not seen the formation of hyaline cartilage, although in 1 case fibrocartilage was

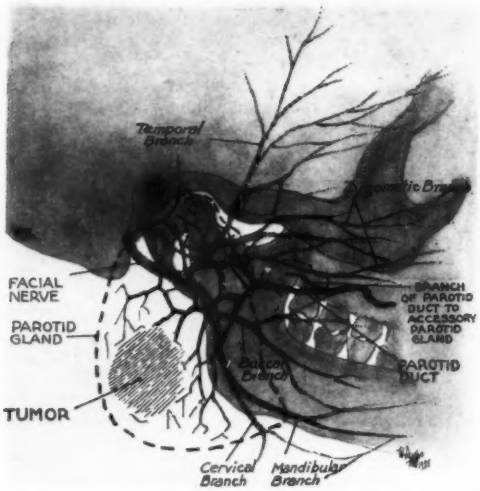


Fig. 6. Diagrammatic drawing of the parotid gland and facial nerve, showing a small tumor.

Fig. 7. Normal parotid duct following the injection of lipiodol.

present. The epithelial elements vary considerably in type, in 2 instances (Cases 73 and 74) being sufficiently true to the normal architecture of the gland as to be designated as actual adenomas. In other instances there are merely groups of cells of epithelial origin, with little or nothing suggesting salivary structures. Stratified squamous epithelium may be present, and epidermoid carcinoma may develop. Case 73 deserves special mention, as there is some question as to whether the lesion is neoplastic or not. This has been studied in detail by Harris, with whose interpretation of the marked proliferation of abnormal duct cells as a tumor we are in accord.

Study of this group leads us to agree with McFarland that the appearance of these mixed tumors is extraordinarily variable and that any attempt to subdivide them on detailed histological or cytological criteria is futile.

The clinical course in histologically divergent tumors is essentially similar. From the clinical course or from examination of the patient, the physician cannot predict whether a largely gland-forming epithelial tumor with well formed, definitely collagenous stroma will be found or a myxomatous mass of tissue with only scattered epithelial cells will be present. In our experience, the tumors in which the epithelial element definitely predominates may give rise only to carcinoma, whereas one rich in myxomatous mesenchymal tissue has given rise to fibrosarcoma (Case 72).

In the 51 mixed tumors, practically every step from predominantly epithelial to predominantly fibroblastic tumors is represented.

Mixed tumors of salivary gland origin are found predominantly in the parotid glands, and in our series 78.4 per cent were found in this location. In the remainder of our cases (21.6 per cent) the tumors were found in the submaxillary gland. They may be found, as mentioned previously, in the sublingual gland and in other locations. Seventy-two and six-tenths per cent of the mixed tumors occurred in women and 27.4 per cent in males. In other groups that have been reported the ratio has been nearly equal. Mixed tumors may occur at nearly any age. Several instances reported in the literature occurred in children, and in our series 7 mixed tumors were found in patients 30 years of age and younger. Three of our patients were more than 60 years of age, and the average age incidence was 44 years. Tumors of this type are usually slow growing and become progressively larger. One of our patients had been aware of the swelling in her neck for 54 years. However, the average duration of growth in this series was 10.7 years. There seems to be very little predilection of these tumors for either side of the neck; in 43.1 per cent of our cases the tumor was on the right side and in 36.9 per cent, on the left.

Fig. 8. Diffuse chronic inflammation of parotid gland—note dilatation of ductules.

Fig. 9. Outline of parotid duct surrounding benign tumor.

Mixed tumors are prone to recur. It has been shown that recurrence may take place many years after operation. McFarland reported 1 case in which the tumor recurred 47 years after removal. In 8 cases of our series the tumor had been previously removed elsewhere, in one of which operation had been performed 30 years before the patient came to the clinic. Recurrence has taken place in only 2 cases of our series to date. In Case 24 of our series the tumor recurred locally with invasion of the mandible 3 years after excision. This patient was treated with large amounts of roentgen-rays and is alive and well at the present time, without evidence of recurrence, 5 years after operation. These tumors occasionally become malignant, however, as occurred in Case 15 of our series.

In 2 patients in our series the tumor was less than 2 centimeters in diameter and had been present for an average of 2.7 years. Forty-nine per cent of the tumors varied in size from 2 to 6 centimeters in diameter and had been present for an average of 9.3 years. Forty-seven per cent of the tumors were more than 6 centimeters in diameter and had been present for an average of 13.1 years. In McFarland's series the incidence of recurrence was greater when the tumor excised was small. As we have already mentioned, so few small tumors were present in our series that data on this point are of no value, but the fact that all

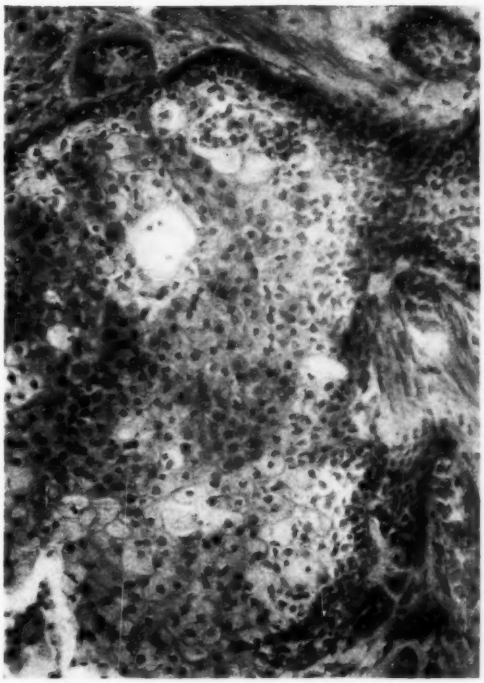

Fig. 10. Epidermoid carcinoma arising in mixed tumors of parotid. +140.

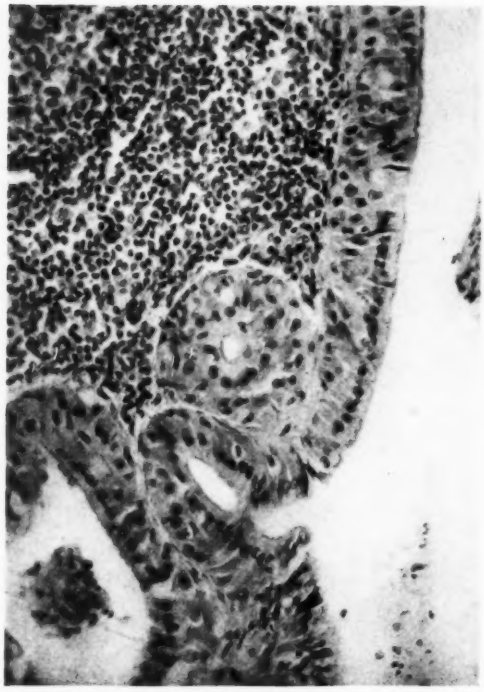

Fig. 11. Adenocystoma lymphomatosum. +445.

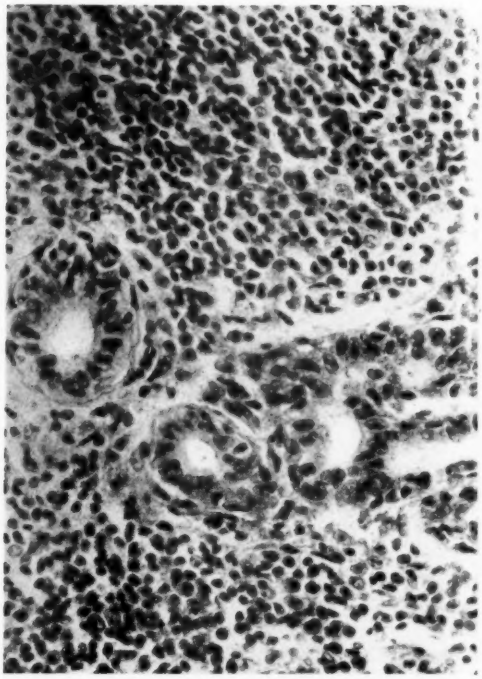

Fig. 12. Chronic inflammation of parotid gland. +365.

but two of the mixed tumors were more than 2 centimeters in diameter at the time of excision may account for the fact that to date recurrence has taken place in only 2 patients. The incidence of recurrence in our series of 51 cases of mixed tumors to date is 4.3 per cent, but we fully realize that this percentage will probably be increased as more time has elapsed. The incidence of recurrence in McFarland's collection of 297 mixed tumors of the salivary gland is 23.23 per cent. If we include in our figures those cases in which recurrence took place before the patient came to the clinic, the incidence is 17.6 per cent.

Two important complications may follow operations for the removal of such tumors. A persistent salivary fistula is a very distressing complication but fortunately it occurs rarely, only once in our series. Paralysis of the facial nerve is a much more common complication (Fig. 6), and if it is of a marked degree it may be distressing to the patient. In this series permanent and complete facial paralysis developed in 5 patients after operation. In addition, a minor and partial degree of facial paralysis occurred after operation in 5 other patients. It usually resulted from injury to the lower branches of the facial nerve, the submaxillary branch, and caused a droop to one side of the lower lip, which completely disappeared after a few weeks. As will be discussed later in this paper, we have never hesitated to sacrifice the facial nerve if we believed, at the time of operation, that the tumor was malignant in character. In 4 of the 5 patients with a permanent facial paralysis, the tumor was recurrent. In these cases of recurrent tumor the technical difficulties of operation are greater because of the scar tissue encountered. Radical excision of the tumor is indicated because the tumor is recurrent and the incidence of injury to the facial nerve is therefore greater than in the uncomplicated cases.

Four cases of papillary adenocystoma were encountered, 1 of which, were it not for similar cells and similar papillary projections in the parotid ductules, might well have been considered as the type of papillary adenocystoma arising from aberrant lateral thyroid tissue. In none of the cases, however, have follicles been discovered nor has colloid been present. In a few instances small papillary projections of similar appearing cells in the ducts have been encountered in otherwise normal salivary glands, and we believe that it is chiefly from the duct epithelium that these tumors develop. We regard these tumors as being potentially malignant, though of a very low grade.

All of the papillary adenocystomas in our series were found in the parotid gland. Three of the patients were women. In 3 cases the tumor was on the left side of the neck. Two of the tumors varied in size from 2 to 6 centimeters in diameter and the 2 remaining were more than 6 centimeters in diameter. All 4 of these tumors occurred in patients 50 years of age or more and varied in duration from 27 months to 25 years. There were no recurrences in this group of cases although one man, aged 71 years, died of an unknown cause 2 years following operation.

In 1 case a tumor closely resembling an adamantinoma was encountered. In fact, in our experience, the most troublesome histological differential diagnosis has been between adamantinomas and aberrant salivary gland tumors. One case of the rare adenocystoma lymphomatosum was encountered, Case 56, which has been previously reported by Harris (7).

Seven cases of cysts of the salivary gland are included in this series. All of these cysts were found in the parotid gland. Five patients were men and 2 were women. In 6 the tumor was on the left side of the neck and in 1 on the right. The duration of the cyst ranged from 2 months to 32 years. The patients were from 41 to 72 years of age; the average age incidence was 56.8 years. All of the cysts at the time of removal were more than 2 centimeters in diameter and 3 were more than 6 centimeters in diameter. Salivary gland cysts are of interest chiefly from a diagnostic standpoint. In several cases the cyst was mistaken for a mixed tumor previous to operation. This study has demonstrated to us also that the incidence of recurrence of these cysts is high if, as was done in several of our cases, excision and drainage only are performed. In 2 of the 6 cases in our series, the tumor which was not completely removed, recurred.

One lesion of the parotid gland which was encountered seven times in this series (Cases 75 to 81) has been of particular interest to us and deserves special emphasis. It is a benign, chronic inflammatory process of the parotid gland. On microscopic examination, diffuse lymphocytic infiltration of the interstitial tissue of the gland is found, with a virtual disappearance of the acinar elements. Frequently the ducts may be barely recognizable, presenting various degrees of metaplasia of their epithelium, in some instances persisting only as cords of keratinized epithelium. This condition is somewhat similar to that encountered in Mikulicz' disease, but is not accompanied, as in that, by involvement of the lacrymal gland, nor is it accompanied by leucemic changes elsewhere, as in the group of cases reported by Thaysen. While these lesions are not properly considered as tumors, nevertheless their importance in differential diagnosis is so great and they are apparently so little recognized that some emphasis of them seems warranted.

Of the 7 cases of chronic inflammation in our series, all were found in the parotid gland. All of the patients were women. Four occurred on the right side of the neck, 1 on the left, and in 2 cases the lesion was bilateral. The duration of this lesion varied from 1 week to 8 years. Four of the patients had had the lesion for less than 1 year, and the average duration in the 7 cases was 22.3 months. The ages of the patients varied from 40 to 72 years, the average age incidence being 55.9 years. At the time of operation all of the tumors were more than 2 centimeters in diameter, 3 varied in size from 2 to 6 centimeters, and 4 were more than 6 centimeters in diameter. Recurrence did not take place in any case of the series. Temporary facial paralysis developed in 1 case following operation, and permanent and complete facial paralysis developed in 3 cases in our series.

TABLE I.—SALIVARY GLAND TUMORS

| Case | Age, years | Sex | Location | | Duration | Size | Date of operation | Recurrence previous to operation | Postoperative complications | End-results |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Gland | Side | | | | | | |
| **Mixed tumors** | | | | | | | | | | |
| 1 | 53 | M | P* | L | 35 yrs. | III | 1- 6-27 | 30 yrs. | 0 | Unknown |
| 2 | 42 | F | P | R | 16 yrs. | III | 11- -27 | 0 | Facial paralysis, permanent | No recurrence in 9 yrs. |
| 3 | 45 | M | P | R | 15 mos. | II | 1-24-28 | 0 | 0 | No recurrence in 8 yrs. |
| 4 | 53 | F | P | R | 9 yrs. | III | 4-11-28 | 0 | 0 | No recurrence in 8 yrs. |
| 5 | 17 | F | S† | R | 2 yrs. | II | 4-30-28 | 0 | 0 | No recurrence in 8 yrs. |
| 6 | 58 | F | P | R | 19 yrs. | II | 1-26-29 | 0 | 0 | No recurrence in 7 yrs. |
| 7 | 53 | F | P | R | 41 yrs. | III | 3- 1-29 | 6 yrs. | 0 | No recurrence in 7 yrs. |
| 8 | 17 | F | P | L | 1½ yrs. | III | 3-29-29 | 0 | 0 | No recurrence in 7 yrs. |
| 9 | 34 | F | P | L | 21 yrs. | II | 6-10-29 | 0 | 0 | No recurrence in 7 yrs. |
| 10 | 21 | F | S | L | 6 yrs. | II | 6-29-29 | 0 | 0 | Unknown |
| 11 | 62 | F | S | L | 10 yrs. | III | 9- 5-29 | 0 | 0 | Unknown |
| 12 | 41 | F | P | R | 3 mos. | II | 9- 9-29 | 0 | 0 | No recurrence in 7 yrs. |
| 13 | 43 | F | S | L | 30 yrs. | III | 2-15-30 | 0 | Facial paralysis, temporary | No recurrence in 7 yrs. |
| 14 | 43 | M | S | L | 1 mo. | II | 5- 3-30 | 0 | 0 | Unknown |
| 15 | 59 | F | P | L | 6 mos. | III | 5-29-30 | 1 yr. | 0 | Recurrence in 3 yrs. (carcinoma) |
| 16 | 44 | F | P | R | | II | 9-29-30 | 0 | 0 | No recurrence in 6 yrs. |
| 17 | 47 | F | S | L | 54 yrs. | II | 10-16-30 | 0 | 0 | No recurrence in 1 yr. |
| 18 | 35 | M | P | L | 16 yrs. | III | 11-21-30 | 13 yrs. | 0 | No recurrence in 3 yrs. |
| 19 | 33 | M | P | L | 4 mos. | II | 1-21-31 | 0 | 0 | No recurrence in 5 yrs. |
| 20 | 47 | F | P | R | 2 yrs. | II | 4-24-31 | 0 | 0 | No recurrence in 4 yrs. |
| 21 | 40 | M | P | L | 15 yrs. | III | 5-13-31 | 0 | 0 | No recurrence in 5 yrs. |
| 22 | 41 | F | S | L | 5 yrs. | III | 6-29-31 | 0 | 0 | Unknown |
| 23 | 60 | F | P | L | 5 yrs. | II | 10-17-31 | 0 | 0 | No recurrence in 5 yrs. |
| 24‡ | 45 | F | P | R | 10 yrs. | II | 10- 3-32 | 0 | 0 | Recurrence in 3 years (local with invasion of mandible) |
| 25 | 60 | F | S | R | | II | 10- 4-32 | 0 | 0 | Killed in 3 wks. |
| 26 | 38 | F | P | L | 10 yrs. | II | 3-27-33 | 0 | 0 | No recurrence in 3 yrs. |
| 27 | 62 | F | P | R | | II | 3-29-33 | 0 | 0 | No recurrence in 3 yrs. |
| 28 | 15 | M | P | L | 4 yrs. | I | 4-18-33 | 0 | 0 | No recurrence in 3 yrs. |
| 29 | 38 | M | P | L | 6 yrs. | II | 4-24-33 | 0 | Facial paralysis, temporary | No recurrence in 3 yrs. |
| 30 | 56 | F | P | R | 9 yrs. | II | 7-10-33 | 0 | 0 | No recurrence in 3 yrs. |
| 31 | 26 | F | P | L | | III | 4-27-34 | 0 | 0 | No recurrence in 2 yrs. |
| 32 | 37 | F | P | L | 17 yrs. | III | 5-12-34 | 17 and 14 yrs. | Facial paralysis, permanent | No recurrence in 2 yrs. |
| 33 | 31 | F | P | R | 18 yrs. | III | 6- 9-34 | 0 | 0 | No recurrence in 2 yrs. |
| 34‡ | 46 | F | P | R | 6 yrs. | III | 8- 8-34 | 6 yrs. | Facial paralysis, permanent | No recurrence in 3 yrs. |
| 35 | 42 | F | P | L | 5 yrs. | II | 9-25-34 | 0 | Facial paralysis, temporary | No recurrence in 3 yrs. |
| 36 | 59 | F | P | R | 28 yrs. | III | 2-25-35 | 0 | 0 | No recurrence in 2 yrs. |
| 37 | 35 | F | P | R | 4 yrs. | III | 3- 5-35 | 0 | 0 | No recurrence in 2 yrs. |

TABLE I.—SALIVARY GLAND TUMORS—Continued

| Case | Age, years | Sex | Location | | Duration | Size | Date of operation | Recurrence previous to operation | Postoperative complications | End-results |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Gland | Side | | | | | | |
| **Mixed tumors** | | | | | | | | | | |
| 38 | 29 | F | P | L | 5 yrs. | III | 7- 8-35 | 3 yrs. | Facial paralysis, permanent | No recurrence in 1 yr. |
| 39 | 54 | M | P | R | | II | 7-10-35 | o | o | No recurrence in 1 yr. |
| 40 | 34 | M | P | L | 1 yr. | III | 9-13-35 | 1 yr. | Facial paralysis, permanent | No recurrence in 1 yr. |
| 41 | 60 | M | P | L | 4 yrs. | III | 9-19-35 | o | o | No recurrence in 1 yr. |
| 42 | 51 | F | P | R | 10 yrs. | II | 11- 8-35 | o | o | No recurrence in 1 yr. |
| 43 | 57 | M | P | R | 20 yrs. | III | 12-11-35 | o | o | No recurrence in 1 yr. |
| 44 | 60 | F | S | L | 1½ yrs. | I | 12-18-35 | o | Facial paralysis, temporary | No recurrence in 1 yr. |
| 45 | 43 | M | P | L | 11 yrs. | II | 2- 6-36 | o | o | No recurrence in 1 yr. |
| 46 | 19 | F | P | L | 3 yrs. | II | 3-18-36 | o | o | No recurrence in 1 yr. |
| 47 | 54 | F | S | L | ? | III | 5-19-36 | o | o | No recurrence in 1 yr. |
| 48 | 45 | F | P | R | 5 yrs. | II | 7-22-36 | o | o | No recurrence in 1 yr. |
| 49 | 50 | M | P | L | 3 yrs. | III | 7-28-36 | o | o | No recurrence in 1 yr. |
| 50 | 42 | F | P | R | 11 yrs. | III | 9-28-36 | o | o | No recurrence in 1 yr. |
| 51 | 66 | F | P | L | 16 yrs. | II | 12-24-36 | o | Facial paralysis, temporary | No recurrence in 1 yr. |
| **Papillary adenocystoma** | | | | | | | | | | |
| 52 | 71 | M | P | L | 25 yrs. | III | 12-14-29 | o | o | Died, 2 yrs, unknown cause |
| 53 | 53 | F | P | R | 10 yrs. | III | 3- 6-30 | o | o | No recurrence in 6 yrs. |
| 54 | 50 | F | P | L | 8 yrs. | II | 8- 1-30 | o | o | No recurrence in 3 yrs. |
| 55§ | 66 | F | P | L | 27 mos. | II | 5-14-31 | 2 yrs. | Facial paralysis, temporary | No recurrence in 3 yrs. |
| **Adenocystoma lymphomatosum** | | | | | | | | | | |
| 56 | 61 | M | P | R | | II | 7-24-35 | o | o | No recurrence in 1 yr. |
| **Cyst** | | | | | | | | | | |
| 57 | 43 | M | P | R | 2 yrs. | II | 11-16-28 | o | o | No recurrence in 8 yrs. |
| 58 | 67 | F | P | L | 2 yrs. | III | 12-14-26 | o | o | No recurrence in 10 yrs. |
| 59 | 45 | M | P | L | 1 yr. | II | 11-20-29 | o | o | Recurrence in 4 yrs. |
| 60 | 61 | F | P | L | 3 yrs. | III | 5- 3-30 | o | o | Unknown |
| 61 | 69 | F | P | L | 2 mos. | II | 12-21-32 | o | o | No recurrence in 2 yrs. |
| 62 | 72 | F | P | L | 32 yrs. | II | 7-13-33 | 12 yrs. | o | No recurrence in 3 yrs. |
| 63 | 41 | F | P | L | 1½ yrs. | III | 10-17-33 | 1 and 2 yrs. | o | Recurrence in 2 yrs. |
| **Papillary adenocarcinoma** | | | | | | | | | | |
| 64 | 68 | F | P | L | 2 yrs. | IV | 9-13-32 | o | o | Died in 5 yrs. (carcinoma of cervix) |
| **Epidermoid carcinoma, I** | | | | | | | | | | |
| 65‡ | 72 | F | P | R | 3 mos. | IV | 10-10-34 | o | Facial paralysis, permanent | No recurrence in 2 yrs. |

TABLE I.—SALIVARY GLAND TUMORS—Concluded

| Case | Age, years | Sex | Location | | Duration | Size | Date of operation | Recurrence previous to operation | Postoperative complications | End-results |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Gland | Side | | | | | | |
| **Epidermoid carcinoma, II** | | | | | | | | | | |
| 66 | 78 | F | P | L | 1 yr. | III | 4–22–31 | 0 | Facial paralysis, permanent | Died in 1½ yrs. (recurrence, local and metastasis) |
| 67 | 36 | F | P | L | 5 yrs. | III | 12–27–32 | 0 | Facial paralysis, permanent | No recurrence in 4 yrs. |
| **Epidermoid carcinoma, III** | | | | | | | | | | |
| 68 | 60 | F | P | R | 20 yrs. | III | 9–27–34 | 0 | 0 | No recurrence in 2 yrs. |
| **Carcinoma, undifferentiated** | | | | | | | | | | |
| 69‡ | 59 | F | P | L | 6 mos. | III | 12–19–31 | 1 yr. | Facial paralysis, permanent | Died in 3 yrs. (recurrence with metastasis) |
| 70¶ | 71 | M | P | R | 6 mos. | III | 10–30–34 | 0 | Facial paralysis, permanent | Died in 2 yrs. (recurrence with metastasis) |
| 71¶ | 49 | F | P | R | 1 mo. | III | 7– 6–34 | 0 | Facial paralysis, permanent | Died in 1 yr. (recurrence with metastasis) |
| **Fibrosarcoma** | | | | | | | | | | |
| 72 | 35 | F | P | L | 3 yrs. | III | 7–19–30 | 3 yrs. | 0 | Died in 3 yrs. (metastasis to liver) |
| **Adenoma** | | | | | | | | | | |
| 73 | 70 | M | P | R | 1 yr. | III | 10– 2–34 | 0 | 0 | No recurrence in 2 yrs. |
| 74 | 63 | F | P | L | 10 yrs. | III | 10–26–36 | 0 | 0 | No recurrence in 1 yr. |
| **Chronic inflammation** | | | | | | | | | | |
| 75 | 40 | F | P | Both | 8 yrs. | IV | 7– 7–28 | 0 | Facial paralysis, permanent | No recurrence in 6 yrs. |
| 76 | 40 | F | P | Both | 6 mos. | III | 12–14–28 | 0 | Facial paralysis, temporary | No recurrence in 6 yrs. |
| 77 | 65 | F | P | R | 1 wk. | II | 1–23–32 | 0 | 0 | No recurrence in 2 yrs. |
| 78 | 46 | F | P | R | 2 yrs. | II | 4–16–32 | 0 | 0 | No recurrence in 1 yr. |
| 79 | 60 | F | P | L | ? | III | 6–20–32 | Approximately 1 yr. | Facial paralysis, permanent | No recurrence in 4 yrs. |
| 80 | 72 | F | P | R | 1 mo. | II | 7– 7–32 | 0 | 0 | No recurrence in 4 yrs. |
| 81 | 68 | F | P | R | 6 mos. | IV | 5– 6–34 | 0 | Facial paralysis, permanent | No recurrence in 2 yrs. |

*Parotid
†Submaxillary
‡Roentgen-ray treatment, III
§Roentgen-ray treatment, II
¶Roentgen-ray treatment, IV

Inflammatory lesions of the parotid gland are frequently associated with calculi in the ducts or obstruction of Stensen's duct from other causes. This group of cases, however, is noteworthy in that no calculi were demonstrable in the ducts of the gland, the ducts were patent to probing and, in certain instances, to the injection of lipiodol (5) (Figs. 7, 8, and 9). As will be seen from the clinical data, this rapidly developing process in patients of advanced years has been difficult to differentiate before operation from the malignant tumors. Radical operations were done in the majority of the 8 cases and, as has been previously men-

tioned, the facial nerve was sacrificed in 3 cases. This chronic inflammatory process almost invariably occurs in the parotid gland. Occasionally, a history of an intermittent swelling of the gland may be obtained. Frequently the process is bilateral and as previously mentioned, this occurred in 2 of our 7 cases. At operation a diffuse process involving the entire gland is found, with no demarcation of normal tissue of the parotid gland as in the case of the mixed tumor. If regional nodes are present, they may suggest the inflammatory nature of this process. However, in our series, palpable nodes were not found in any of the cases.

As we have previously stated, the differentiation of this chronic, benign inflammatory process from malignancy has been difficult. We believe, however, that with a greater appreciation of its frequency, when at operation diffuse processes are encountered involving the entire parotid gland, biopsy may be of aid. At times the differentiation microscopically between these inflammatory lesions and malignancy of these glands will be difficult without an extensive experience in the microscopic diagnosis of these tumors.

There were 9 malignant tumors in this group and all were found in the parotid gland. The criteria for malignancy in the case of salivary gland tumors are difficult to establish because of the divergent histological picture. The mixed tumor often lacks differentiation of various of its cells, and there may be poor encapsulation. It would be futile to classify as malignant all those tumors that show evidence of capsule invasion. We have considered only those tumors malignant in which there were combined cellular anaplasia or metaplasia, invasive properties, and evidence of rapid growth which would warrant making a diagnosis of malignancy in whatever tissue these changes might make their appearance. Invasive growth alone, although ordinarily a helpful diagnostic point in the case of malignant tumors, is valueless in the group of salivary gland tumors.

We have never seen a mixed tumor of the parotid gland metastasize as such. Rather, the same situation occurs as that encountered in the case of teratomas of the testis or ovary, in which some one element undergoes malignant change and metastasizes as a specific cell type, so that it would be impossible to hypothecate from the metastasis alone the teratoid nature of the tumor. On this basis there were 9 cases of malignancy; 1 low grade papillary adenocarcinoma; 4 epidermoid carcinomas, 2 of grade II, and 1 each of grades I and III; 3 undifferentiated carcinomas, and 1 fibrosarcoma developing from a mixed tumor.

Case 15 is of considerable interest. The first specimen of this tumor showed a characteristic mixed tumor with myxomatous stroma but no evidence of malignancy. A year and a half after removal, recurrence took place which appeared as a relatively undifferentiated tumor of somewhat uncertain cell type. The diagnosis was originally in question, but it was probably a carcinoma in spite of the fusiform character of many of the cells. In spite of intensive roentgen-ray treatment, death resulted a year and a half later.

In contrast to this is Case 72. Only fairly well developed fibroblasts with numerous mitotic figures were found in the tumor at the first operation. Since this tumor had been present for 3 years it is probable that it originated in a mixed tumor of the parotid gland, although there was no definite evidence of the existence of a mixed tumor at the time of operation. Three years later the metastatic lesion in the liver showed it to be a definite fibrosarcoma, essentially similar in histological appearance to the first specimen obtained from the parotid gland.

Eight of the 9 patients with malignant tumors were women. The average age, 59.8 years, is considerably greater than the average age of the patients who had mixed tumors (44 years), although apparently they may occur at nearly any age. In our group the patients varied from 36 to 78 years. The average duration of these tumors was also much less than the duration in the mixed tumor group, being 3.6 years as compared to 10.7 years in the latter. In 4 of the malignant cases, swelling had been present for less than 1 year but 1 patient had been aware of a tumor for 20 years. The majority of the malignant tumors are of short duration and rapidly growing but they may be slow growing tu-

mors. All of the malignant tumors in our series were 7.5 by 5.5 centimeters or larger at the time of operation.

The only patient in our series with papillary adenocarcinoma was apparently cured but died at the end of 5 years with a carcinoma of the cervix. One patient with epidermoid carcinoma, grade I, and 1 with an epidermoid carcinoma, grade II, are alive and well 2 and 4 years, respectively, after operation. One patient who had had an epidermoid carcinoma, grade III, removed was alive and well at the end of 2 years. All of the patients who had an undifferentiated carcinoma and the patient with fibrosarcoma were dead within 3 years after operation. Radical excision was done in this group of cases as shown by the fact that the facial nerve was sacrificed in 6 of the 9 patients. Supplementary roentgen or radium therapy, after operation, was employed in 4 of the cases. Two of the 9 malignant tumors had been excised previously and had recurred before the patient came to the clinic.

It has been the policy in this clinic to advise excision of tumors of the salivary gland, particularly tumors that are increasing in size. McFarland (9) has emphasized the high incidence of recurrence in the mixed tumor group following operation on small tumors. Our experience with the small salivary gland tumors has been so limited that we cannot comment on this attitude. Complete excision of tumors without leaving particles of tumor tissue behind has frequently, in our experience, been more difficult technically in the small than in the large tumors, and this may be the explanation for the reported high incidence of recurrence in the smaller salivary gland tumors. Too much emphasis cannot be placed on the importance of careful complete excision of these tumors without rupture of the capsule when present, and spilling of the tumor contents. Fortunately the majority of these tumors lie above the main branches of the facial nerve, but, as we have previously stated, when malignancy is suspected and it is impossible to remove the tumor entirely without sacrificing the nerve this should be done. Complete and permanent paralysis of the facial nerve has in the past been a very disturbing deformity and the various plastic procedures that have been developed for its correction have not been particularly successful. Recently, anastomosis of the spinal accessory nerve and the hypoglossal nerve to the peripheral end of the facial nerve and resection of the superior cervical sympathetic ganglion have offered promising results. If at the time of operation a diffuse process involving the entire gland is found, if there is a history of intermittent swelling of the tumor, or if it is bilateral, before the facial nerve is sacrificed the presence of an inflammatory process should be suspected and biopsy should be performed, with careful microscopic study by a pathologist who has had considerable experience in the diagnosis of salivary gland tumors.

The effect of roentgen-rays and radium in our group of patients has not been encouraging with the exception of the one case in which mandibular invasion had occurred. We fully realize, however, that in the past the dosage given probably was inadequate, and we hope in the future that with increasing experience with roentgen-ray and radium therapy, the results may be improved. The continuation of large doses of deep roentgen-rays supplementary to radical excision in patients with malignant tumors should be continued until it is definitely proved that such treatment is of no value.

In the benign tumors of salivary gland origin with careful complete excision, we believe that the incidence of recurrence will not be high. However, the fact that these tumors may recur many years after operation must be realized. We do not agree with the premise that the outlook for patients with malignant tumors of the parotid gland is hopeless. With improved diagnostic criteria, with radical excision, and with improved roentgen-ray therapy, better end results may be anticipated. It must be remembered that it may be necessary to sacrifice the facial nerve in many of these patients if a cure is to be obtained. It has always been the policy in this clinic to explain carefully to patients with parotid tumors of any type previous to operation that facial paralysis may result after operation if the tumor is so extensive that it is impossible for it to be removed without causing injury to the nerve.

SUMMARY

1. Eighty-one tumors of salivary gland origin are reported.

2. From this series of cases the incidence is definitely in favor of the embryologic misplacement of cells as the mode of origin of most salivary gland neoplasms.

3. The extremely variable histological appearance of the mixed tumors is discussed and clinical data of the 51 mixed tumors are presented.

4. The incidence of recurrence of the mixed tumors following operation at the clinic is 4.3 per cent.

5. Papillary adenocystoma, cysts, the rare adenocystoma lymphomatosum, and the uncommon adenomas are discussed.

6. Our criteria for diagnosis of malignant tumors of the salivary gland are presented and the cases of 9 patients, 3 of whom are alive and well at the present time, are reported.

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# THE ACUTE SAFETY OF ETHER, DIVINYL ETHER, AND CHLOROFORM IN THE PRODUCTION OF THE "OBSTETRIC DEGREE" OF ANALGESIA

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IT is a most remarkable circumstance that, after 90 years of constant progress in chemistry and pharmacology, ether still remains our safest and most generally useful anesthetic. None of its newer rivals is capable of entirely replacing it. Even in those clinics in which the preference is given to some other anesthetic, ether is usually held in reserve for those more or less frequent instances in which the favored agent fails to provide satisfactory anesthesia.

There is no doubt, however, that the action of ether leaves much to be desired. Particularly objectionable are the discomfort it causes the patient and its injurious effects upon the metabolism, kidney function, and respiratory epithelium. Most of the substitutes for ether which are now in use owe their vogue to their alleged superiority on one or more of these points.

For the reasons just given, we must welcome the introduction of a new anesthetic agent, but such an agent ought to be subjected to a most searching experimental and clinical study before it is finally accepted as superior to our present anesthetics. The high degree of efficiency of the agents now in use make such an evaluation a complex and difficult problem. It is a task which cannot be done hurriedly.

We were led to undertake the following studies by the report of Knoefel, Guedel and Leake, 1931, that divinyl ether or "vinethene"[1] ($CH_2$:CHOCH:$CH_2$) showed great promise of qualifying as a useful general anesthetic. We were especially interested in the claim that anesthesia was induced rapidly and pleasantly and was not followed by nausea. It is obvious that an anesthetic having these properties might be of great value in obstetrics. The drug has already attained a certain vogue for this purpose (*2*, *3*).

In the experiments we are reporting, we have been concerned with the determination, in dogs, of the acute safety of vinethene analgesia under the conditions obtaining in obstetrics, dentistry, and minor surgery, and its comparison with that of the older agents, chloroform and ether. We have included, in addition to the conventional determination of the so called margin of safety as expressed by the percentile relationship of the lethal to the effective dose, a statistical study of resuscitation from an overdose of these various agents.

## TECHNIQUE OF THE DETERMINATION OF THE SAFETY OF AN ANESTHETIC

The acute safety of an anesthetic may be studied upon both human and laboratory material.

*1. Clinical determination.* The clinical determination of the acute safety consists chiefly in a statistical study of the anesthetic mortality in a series of administrations. This is by no means as simple a procedure as one might expect. In fact, an analysis of the various published statistics shows a marked lack of agreement in the percentage mortality attributed to even our oldest anesthetics, although our experience with these now covers many millions of administrations. The reader, therefore, who sets out to make a comparison of the clinical mortality from the use of a new anesthetic with that from one of the older anesthetics is up against an extremely difficult, if not impossible, task. This very unsatisfactory state of affairs may be expected to continue until detailed reports of all anesthetic fatalities are compulsory and a standardized definition of the term "anesthetic death" is adopted by all workers in this field.

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[1] Vinethene supplied by Merck and Company.

Our own guess (and it is only a guess) is that the mortality of ether anesthesia is somewhere between 1:2,000 and 1:3,000. This is considerably higher than is indicated by most statistics, such as those quoted by Sollman, which range from 1:5,100 to 1:23,200 and average 1:16,000, but is, on the other hand, considerably lower than certain of the recent compilations such as those of Bartlett and Simmons, 1932, who encountered an acute ether mortality of 1:738 in the course of 10,325 gynecologic abdominal operations upon good risk patients and of Rollison, 1930, who reported a mortality of 1:1,010 in 6,062 general anesthesias of various types. It is quite evident, therefore, that the published statistics of anesthetic mortality do not provide a sound basis for the comparative clinical evaluation of a new drug.

If, as a basis for discussion, we take 1:3,000 as the expected mortality in ether anesthesia, it is at once apparent that we have in ether an agent which, in spite of its admitted defects, is none the less a remarkably safe anesthetic. Certainly, we are not justified in lightly abandoning it in favor of some new substance which has had as yet only the most superficial investigation.

A great deal of unnecessary confusion would be avoided if investigators sufficiently realized that statistical studies of anesthetic mortality require data from a very large series of administrations. The dangerous fallacy of attempting to determine an "average" from an inadequate series is well known to all bioassayists. We believe that this same principle applies with equal force to the closely related field of mortality statistics in anesthesia. That this obvious fact is not widely appreciated is sufficiently illustrated by the numerous prematurely enthusiastic reports which have accompanied the introduction of so many of the new anesthetics.

2. *Laboratory determination.* The laboratory worker enjoys a great advantage over the clinical observer in that he can deliberately induce death in his experimental animal under a standardized set of conditions. He is thus enabled to observe at will, under controlled conditions, the effects of measured toxic doses of an anesthetic. Data of this type are impossible to obtain under clinical conditions. It should, however, never be forgotten that information obtained from laboratory animals is valid only for that particular species and that, although such information may serve as a useful clinical guide, it cannot take the place of prolonged and accurate clinical observation.

As the following brief exposition will show, the members of the inhalation group of anesthetics have in common certain pharmacological peculiarities which make the investigation of their margin of safety more difficult than a similar study upon non-volatile anesthetics. Fortunately for the investigator working with this latter group of drugs, the methods of administration usually employed, particularly the intravenous, preclude any serious irregularities in the rate of absorption. Therefore, ordinarily it may be assumed that the amount of the drug entering the animal's blood stream is identical with the amount administered. Furthermore, the rate of detoxification and excretion of these drugs is usually so slow, compared with the rate of absorption, that it also may be assumed that the amount of the drug retained within the animal, i.e., the amount responsible for the narcosis is, for all practical purposes, identical with the amount administered. This definite relationship existing between the amount of a non-volatile anesthetic which is administered and the degree of the narcosis which subsequently develops greatly simplifies the task of the investigator.

With the members of the group of volatile anesthetics, however, the experimental conditions are quite different. These drugs are necessarily administered in the gaseous state and the amount entering the blood stream is, therefore, not easily ascertained. The excretion of these substances, in contrast to that of the members of the non-volatile group, is so rapid that, unless special precautions are taken, the amount retained in the animal, i.e., the amount actually responsible for the narcosis, bears only an indirect relationship to the amount administered.

The peculiar experimental conditions noted have compelled most of the previous investigators to direct their studies to the relation-

ship which they have supposed to exist between the concentration of the anesthetic in the inspired air, blood, and tissues, and the depth of narcosis. But, in the belief of Haggard, no such relationship exists until after full equilibrium has been established because, "Only at full equilibrium for any tension of ether inhaled can the physiological state (narcosis) be correctly correlated with the inspired or expired ether concentration or with that of the blood drawn from any source" (6). As reference to the following authorities will show, there are substantial reasons for doubting that these equilibria are ever reached during the relatively short period of clinical anesthesia (7, 10). These equilibratory relationships, consequently, are complex and partake of the nature of a curve the shape of which is determined, in part, by the involved time factor. It follows from these considerations that the margin of safety of a given inhalation anesthetic may vary significantly with the duration of the anesthesia (7). Since most of the margins of safety found in the literature have been calculated from data obtained during very prolonged administrations, they cannot be considered as necessarily measuring the safety of an anesthetic when used for very brief periods. It is obvious that experiments designed to make this determination with particular reference to obstetrics, dentistry, and minor surgery, should involve anesthesias which are comparably brief. But as far as we have been able to ascertain, none of the published studies has been so planned and their significance for these specialized fields is, therefore, questionable.

## DESCRIPTION OF EXPERIMENTAL PROCEDURE

Our primary object has been to determine the relative acute safety of ether, chloroform, and divinyl ether when used to produce the degree of analgesia required in obstetrics, dentistry, and minor surgery. Since the determination of the margin of safety of an anesthetic is, in essence, a biological assay, it should be conducted, as far as possible, in accordance with the established principles governing this procedure. We have, therefore, tried to plan our experiments to conform with the more important of these principles. Among such are:

1. *Number and character of the individuals making up the series.* To the bio-assayist an adequate series is one which is numerically large enough to furnish a median dose which would not be significantly changed by the addition to the series of data from any number of other individuals. The exact number of animals thus required for an adequate series is an individual matter for each drug and can be determined only after a study of the curve of response of a large population to that drug. Since a series of this magnitude for each of the three anesthetics was, from considerations of time and expense, beyond our resources, we are presenting, for each drug, data derived from only 35 dogs. Experience, however, has shown that a series of this size is adequate for the bio-assay of most drugs. A larger series would have necessitated the use of smaller animals such as mice or rats. In our opinion the greater physiological resemblance existing between dogs and men, together with the greater ease of individualizing experiments upon dogs, more than compensates for the loss in the number of individuals which the use of these animals has necessarily entailed. Mongrel dogs in good health kept on a diet of Purina Chow were used. Of these approximately two-thirds were males.

2. *The experimental technique employed should be so standardized as to make possible a direct comparison between the data from any one experiment, animal or drug with that from any other experiment, animal or drug in the series.* The importance of employing a rigidly standardized technique in all the experiments of a series of this character cannot be overemphasized. This is a fundamental principle of biological assay and, indeed, it is not too much to affirm that in its absence no assay has been accomplished and that, therefore, the toxicity of the drug in question has not been determined. Haphazard experimentation is responsible for much of the confusion which exists in pharmacology today and perhaps in no field is this more evident than in that of the inhalation anesthetics. Most of the experiments found in the literature upon the margin of safety of the various inhalation anesthetics are of this haphazard nature and consequently they do not provide a very sound ground for the

determination of their comparative merits, nor do they serve as a satisfactory basis for the evaluation of a proposed new anesthetic.

In an attempt to meet this obvious objection to much of the older work we have conducted all of our experiments according to the following routine.

Figure 1 is a diagrammatic sketch of the apparatus employed. It is essentially a closed soda-lime absorption system within which the anesthetic is volatilized in an atmosphere of oxygen. The use in this way of the Waters technique has enabled us to keep the dead space of the apparatus to within 5 to 6 liters and to imitate this popular clinical method closely in our experiments. The metal mask contains a rubber diaphragm through which, by means of a syringe and needle, the measured dose of the anesthetic is injected on to a wire screen for volatilization. The mask is fitted tightly to the dog's muzzle by means of a rubber face piece. Every effort is made to prevent gas leakage at this point and the entire system is tested frequently for leaks. Since the screen is warmed by the animal's exhalations, volatilization of the anesthetic is extremely rapid and the drug develops its maximum effects within an average period of 2 minutes. The interior of the mask is examined frequently at the end of experiments in order to determine the completeness of volatilization. At no time did we find more than 6 per cent of the administered drug remaining unvolatilized. In these cases the non-volatilization was due to the solution of a portion of the drug in the saliva secreted into the mask. The presence of an excess of oxygen precluded the possibility of asphyxia playing any part in the analgesia.

Each dog was subjected to two series of experiments with each of the 3 anesthetics. In the first series the minimal dose for analgesia (M.D.A.) was determined. This was taken as the smallest dose which produced insensitivity, during at least two out of three successive experiments, to a strong, faradic, electrical stimulus applied to the skin of the lower abdomen. The stimulus was furnished by an inductorium kept at a constant setting throughout the entire series. The degree of anesthesia thus secured was somewhat more profound

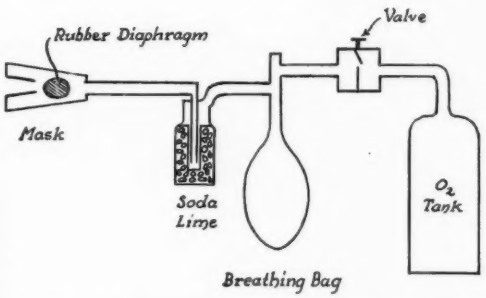


Fig. 1. Diagrammatic sketch of apparatus used.

than is ordinarily required during the first part of the second stage of labor but, of course, was very much lighter than ordinary surgical anesthesia. The initial dose of the anesthetic administered to an animal was usually somewhat smaller than the average required for analgesia. Successive doses were then varied from the preceding by 10 per cent. We mean, therefore, by the minimal dose for analgesia, the amount of the drug which produced analgesia in at least 2 of 3 consecutive experiments and which, when reduced by 10 per cent, produced analgesia in not more than 1 of 3 consecutive experiments. Consequently, the establishment of the minimal dose for analgesia for any given animal was not the result of a single experiment, but was repeatedly confirmed by the use of doses 10 per cent both above and below this figure. It follows from the above that the further the minimal dose for analgesia of a particular animal diverged from the median, the more thoroughly was this figure established by means of repeated experiment.

In the second series of experiments, the minimal dose of each drug required to produce an apparently permanent respiratory arrest (M.D.R.A.) was established. For this purpose the initial dose employed was usually below the average. It was then increased or decreased by 10 per cent as required, until we had determined an amount of the drug which produced an apparently permanent respiratory arrest and which when reduced by 10 per cent failed to cause arrest. After the respiratory paralysis had lasted 15 to 20 seconds, the mask was removed and the animal was

resuscitated by artificial respiration and the administration of oxygen. In order to save the animal as much anesthesia as possible, a single respiratory arrest was taken as the end point of these experiments. In the case, however, of a minimal dose for respiratory arrest which diverged greatly from the median, the use of doses varying by only 10 per cent served repeatedly to confirm the unusual response of the animal. A period of from 2 to 3 days intervened between experiments on individual dogs.

The use of the same dog for a succession of anesthesias obviously raises the question of tolerance. For the following reasons we do not believe that the development of tolerance complicates our results to a significant degree: (a) In order to avoid weighting of the series by any possible cross tolerance, the animals were roughly divided into three groups, each of which began the series with a different anesthetic. (b) In a number of animals the experiments were interrupted for periods of one month or more. Upon resumption of the experiments no constant change was seen in their susceptibility. (c) Finally, it is scarcely conceivable that a series of anesthesias, amounting in sum to the equivalent of only 60 minutes of ordinary anesthesia, could, when spread over a period of 3 months, result in an important degree of tolerance.

With the technique we have described an undetermined part of the administered dose remained unabsorbed from the dead space of the apparatus. Therefore, that part of the administered dose which remained unabsorbed at the conclusion of the experiment should be deducted from our figures, in order to arrive at the actual amount of the drug acting. To have obtained this information would have required an analysis of the contents of the dead space at the conclusion of each of the 1,103 experiments. If we had attempted this laborious procedure, we would necessarily have been compelled to reduce the number of experiments and, whatever advantage the series might have gained in accuracy, would have been nullified by a reduction in the number of individuals.

*3. The personal element in work of this character is very great. For this reason the bio-assayist is accustomed to carry out his own "control" experiments concurrently with each assay series.*

Experience has shown that, even when the same technique is used, the experimental results obtained by different workers may vary considerably. Consequently, whenever a determination of the comparative toxicity of two drugs is attempted, the chances of error are increased unless *both* of the drugs are studied by the same investigator and by the same technique.

As a general rule the workers who have studied the toxicity of the inhalation anesthetics have not standardized their individual experiments and unfortunately, have limited themselves to the investigation of only one drug. This, together with the confusion resulting from the use by one worker of a technique which differs in essentials from that of other workers, gives the available data in this field such a heterogeneous character that it is difficult to use them as a basis for the comparative evaluation of two or more anesthetics.

In order to facilitate such a comparison we have added to our investigation of divinyl ether a similar study, by the same technique, of the older anesthetics, ether and chloroform.

*4. It is desirable that the experiments be planned, whenever possible, to yield data in terms of a measured amount of the drug per unit weight of animal.* It has been the almost universal custom for those investigating the margin of safety of the non-volatile anesthetics to report their findings in terms of a measured dose of the drug per unit of body weight and for those investigating the inhalation anesthetics to report in terms of the volumes per cent in the inspired air which have been allowed to act over varying periods of time, etc. A direct comparison of the acute safety of the various inhalation anesthetics with those of the non-volatile group for this reason cannot be made on the basis of the published data. In view of the fact that the anesthetist is often called upon to choose between these two types of anesthetic agents, this comparison would seem to be highly desirable. Quite aside from the above considerations there seems to be little doubt that, when the quantity of the drug acting is expressed in terms of the measured amount per unit of

TABLE I.—COMPARATIVE POTENCIES AND MARGINS OF SAFETY

| | Potency | Margin of safety |
|---|---|---|
| Ether | 100 | 100 |
| Divinyl ether | 186 | 70 |
| Chloroform | 722 | 63 |

body weight, it is more readily understood by most laboratory workers and clinicians than when expressed as volumes per cent, etc.

5. *An assay of the acute toxicity of an inhalation anesthetic should also include a systematic study of the ease and certainty of resuscitation from an overdose.* Although studies on this aspect of the toxicity of drugs are not a usual part of biological assays, it is obvious that the information so obtained would be of practical value. Its importance, in the case of the inhalation anesthetics, has already been briefly discussed. As a general rule, a patient can be more readily resuscitated from an accidental overdose of one of the inhalation anesthetics than from a similar overdose of a non-volatile anesthetic. This important property of the volatile anesthetics makes these drugs much safer in practice than a mere consideration of their margin of safety would indicate.

## DISCUSSION OF RESULTS

Our experimental results, after giving to ether the arbitrary value of 100, are briefly summarized in Table I. This table shows that chloroform has the highest potency but the lowest margin of safety and that ether, the least potent of the 3 has the highest margin of safety. Divinyl ether occupies an intermediate position with respect to both these properties.

From Table II it may be seen that our animals exhibited a quite considerable variation in susceptibility to all three of these drugs. The margin of safety was calculated from the formula $\frac{\text{M.D.R.A.} - \text{M.D.A.}}{\text{M.D.A.}}$ expressed as per cent. Dosage is given in cubic centimeters per kilogram of body weight.

M.D.A., minimal dose for analgesia.
M.D.R.A., minimal dose for arrest of respiration.

A spread of the magnitude we have encountered, between the least and most susceptible of the series, may be explained as being (1) only apparent and due, therefore, to errors in technique, or (2) as reflecting an actual variation in susceptibility. A certain degree of error is inherent in work of this character, but as we have mentioned above, it follows as a consequence of the technique we have employed, i.e., the use of repeated doses varying by only 10 per cent, that the further the response of an animal diverged from the median, the more frequently was the response confirmed. Therefore, it is just those abnormal responses, which might be taken as pointing to

TABLE II.—SUSCEPTIBILITY TO DRUGS

| | | Ether | Divinyl | Chloroform |
|---|---|---|---|---|
| Experiments | | 389 | 339 | 375 |
| M.D.A. | minimum | 0.39 | 0.19 | 0.058 |
| | maximum | 1.10 | 0.46 | 0.132 |
| | median | 0.65 | 0.35 | 0.090 |
| M.D.R.A. | minimum | 1.00 | 0.50 | 0.110 |
| | maximum | 3.30 | 1.05 | 0.255 |
| | median | 1.65 | 0.70 | 0.175 |
| Margin of safety | minimum | 64% | 39% | 31% |
| | maximum | 340% | 284% | 219% |
| | median | 144% | 100% | 90% |

Total experiments in series, 1,103.
M.D.A., minimal dose for analgesia.
M.D.R.A., minimal dose for arrest of respiration.

TABLE III.—MINIMAL DOSE

| Analgesia | | | Respiratory arrest | | |
|---|---|---|---|---|---|
| Dog A.M., female, weight 6.75 kilograms. Anesthetic, divinyl ether | | | Dog A.I., male, weight 8.1 kilograms. Anesthetic, divinyl ether | | |
| Dosage in c.cm. per kilogram (median dose of series is 0.35) | | | Dosage in c.cm. per kilogram (median dose of series is 0.70) | | |
| Exp. No. | | | Exp. No. | | |
| 1. | 0.30 | Analgesia | 1. | 0.72 | Respiratory arrest |
| 2. | 0.30 | Analgesia | 2. | 0.63 | Respiratory arrest |
| 3. | 0.27 | Analgesia | 3. | 0.56 | Respiratory arrest |
| 4. | 0.27 | Analgesia | 4. | 0.50* | Respiratory arrest |
| 5. | 0.24 | Analgesia | 5. | 0.45 | Non-arrest |
| 6. | 0.24 | Analgesia | | | |
| 7. | 0.24 | Analgesia | | | |
| 8. | 0.215 | Analgesia | | | |
| 9. | 0.215 | Analgesia | | | |
| 10. | 0.19* | Analgesia | | | |
| 11. | 0.19* | Analgesia | | | |
| 12. | 0.17 | Incomplete | | | |
| 13. | 0.17 | Incomplete | | | |
| 14. | 0.17 | Incomplete | | | |

*Lowest doses encountered in the divinyl ether series.

TABLE IV.—CORRESPONDENCE OF DOSAGES

| | M.D.A. in c.cm./kgm. | | | M.D.R.A. in c.cm/kgm | | |
|---|---|---|---|---|---|---|
| | Average | Median | Difference | Average | Median | Difference |
| Ether | 0.67 | 0.65 | 3.1% | 1.73 | 1.65 | 4.8% |
| Divinyl ether | 0.33 | 0.35 | 5.7% | 0.69 | 0.70 | 1.4% |
| Chloroform | 0.091 | 0.090 | 1.1% | 0.177 | 0.175 | 1.1% |

errors in technique, which have been the most carefully established. This will be made clear by reference to Table III, in which the experiments establishing the lowest values obtained for the minimal dose of divinyl ether for analgesia and minimal dose for respiratory arrest are presented in detail.

To a very limited extent the accuracy of an assay may be inferred from the tendency toward coincidence of the average dose of the whole series and the median or amount effective in 50 per cent of the animals (12). The extent to which these doses correspond in our series is shown in Table IV.

Graphs constructed from the data obtained from each of these three drugs all show the S shaped curve regarded as characteristic of drug toxicity determinations. They are not reproduced here because of space limitations. Fairly steep symmetrical curves were obtained with both chloroform and divinyl ether. The ether curves, however, reveal a relatively greater degree of skew. This may be taken as indicating that of the three drugs, ether is probably the least predictable and is, therefore, the least reliable when administered by methods involving predetermined dosage. The greater margin of safety of ether, however, undoubtedly more than compensates for this disadvantage.

Although the individual variation in the response observed in our dogs is large, it is probably no greater than has very often been encountered during similar studies in both animals and man. "Even when all these precautions (ancestry, age, diet, weight, and sex) are taken animals still show a wide variation in their response to drugs," Clark, 1937. Poe, Suchy, and Witt, 1936, who measured the lethal dose of strychnine base for 4,000 rats, found that, in the case of adult male rats, the mean lethal dose was 2.25 milligrams per kilogram. However, 1 in 25 of their rats was killed by 1.5 milligrams per kilogram and a similar number survived 3.5 milligrams per kilogram. McCollum, 1930, found that the anesthetic concentration of chloroform varied in 5 dogs from 19 to 38 milligrams per 100 cubic centimeters of blood. In the case of evipal, McNelis, 1936, found that in 1,000 cases, one man required 4 grams to produce anesthesia and that, with one woman, 0.9 gram appeared to be an overdose. Working with divinyl ether, Goldschmidt and associates, 1934, found that the anesthetic concentration in 8 human subjects varied from 11.3 to 22.8 milligrams per 100 cubic centimeters of blood and that, in 20 dogs, this concentration ranged from 18 to 47 milligrams per 100 cubic centimeters of blood. These few examples selected at random from the literature illustrate the well established principle that an individual variation in the response to all drugs is inherent in all biological material. Therefore, although extensive studies may establish beyond doubt the response of the average of a population, that of any of its individual components must always remain unpredictable and instances of extreme susceptibility, often erroneously ascribed to idiosyncrasy, are mathematically certain to occur in every large series of drug administrations. The anesthetics are certainly no exception to this rule and the conclusion is inescapable that the production of a uniform response such as surgical anesthesia is safe only in so far as the dose is adjusted to the susceptibility of the individual. The inhalation method of anesthesia, therefore, must be regarded as fundamentally safer than the method of predetermined dosage.

It is self-evident that an anesthetic death may be regarded as also a failure of resuscitation. If resuscitation from an overdose of an anesthetic were always possible, there need never be an anesthetic death. This is an aspect of the toxicity of anesthetics which appears to have been largely neglected by the previous workers in this field. And yet, it is obvious that the margin of safety alone is an inadequate guide to the probable clinical mortality which will accompany the use of a particular anesthetic for the reason that the acute mor-

tality is determined not only by the margin of safety, but also by the ease and certainty of resuscitation from accidental overdose. After all, the margin of safety is concerned only with the likelihood of an overdose. The likelihood of *death*, however, in the event of overdose, depends entirely upon the probability of resuscitation. The ease and the certainty of resuscitation from overdosage are a phase of anesthetic toxicity which depends upon pharmacologic considerations entirely separate from those responsible for the margin of safety and is possessed by the individual anesthetics to a varying degree. In practice it is probably of even greater importance as the determining factor of anesthetic death. This is brought out in Table V which summarizes the data obtained during the resuscitations from 136 instances of overdosage with the three anesthetics. It will be seen that the probability of resuscitation from an overdose of chloroform is very much less than that from both ether and divinyl ether.

The point we have emphasized above, namely, that the margin of safety alone is a quite inadequate guide to the probable clinical mortality, is made clear when the position of divinyl ether with respect to the likelihood of an overdose (Table I) is contrasted with respect to the probability of resuscitation following an overdose (Table V). Although in our hands the margin of safety of chloroform is only 10 per cent below that of divinyl ether, it should, because of its marked inferiority with respect to resuscitability, prove to be much the more dangerous drug.

Our resuscitations from both ether and divinyl ether overdosage have been uniformly successful. There are two points, however, concerning the behavior of our animals after an overdose of these drugs, which deserve some comment. It was noticeable that the animals poisoned with divinyl ether tended to recover more rapidly under artificial respiration than those poisoned with ether. This may be attributed to the relatively rapid elimination of divinyl ether. Rapid elimination of a poison is a most important factor in successful resuscitation and, in this respect, divinyl ether appears to be definitely superior to ether. On the other hand, the circulatory condition of the animals at the time of respiratory arrest, as judged by the degree of cyanosis, strength of heart beat, etc., appeared to be considerably better following an overdose of ether than of divinyl ether. With respect, therefore, to its circulatory toxicity—a factor of equal importance in resuscitation—divinyl ether appears to be definitely inferior to ether.

TABLE V.—STUDY OF RESUSCITATION FROM RESPIRATORY ARREST

| | No. of respiratory arrests | No. of failures to resuscitate | % of failure to resuscitate |
|---|---|---|---|
| Ether | 42 | 0 | 00 |
| Divinyl ether | 45 | 0 | 00 |
| Chloroform | 49 | 6 | 12.2 |

If we regard resuscitation from poisoning by an inhalation anesthetic as essentially a race between the elimination of the toxic agent and the development of its fatal effects upon the circulation, it is evident that each of these drugs possesses an important advantage over the other. We are inclined to believe, however, that ether, due to its greater margin of safety, will prove to be the safer of the two drugs. During the course of our experiments it became quite evident that chloroform, with regard to both the rapidity of its elimination and the condition of the circulation at the time of respiratory arrest, was inferior to both ether and divinyl ether. The disastrous effect of a combination of slow elimination and high toxicity to the circulatory system is reflected in the high percentage of failures to resuscitate from chloroform overdosage which is shown in Table V.

There is another aspect of the behavior of our dogs during chloroform poisoning which is of interest. Although, during the course of our experiments, we administered 121 toxic doses of chloroform by a very brusque method —a method which violated all the established principles of safe chloroform administration—we encountered but one instance of primary chloroform collapse. In all of our other experiments the animals proceeded to the condition of bulbar depression or paralysis through the regular sequence of the stages of anesthesia. This is in remarkable contrast to the frequent cardiac collapse encountered when chloroform

is very rapidly and carelessly administered to dogs by the conventional drop method. We attribute the relative absence of chloroform collapse in our series to the use of pure oxygen instead of air as a volatilizing medium. Evidently the excess of oxygen served to protect the heart from the usual toxic effects of chloroform.

A few general comments may be made on the response of our animals to these anesthetics. We are unable to confirm the observations of Leake, Knoefel, and Guedel, 1933, that less salivation and mucus flow occurs during the administration of divinyl ether than of ether. In our experience divinyl ether produced more salivation and mucus flow than ether. The same authors found that divinyl ether was followed by less postanesthetic nausea and vomiting. Our dogs showed postanesthetic nausea and vomiting so infrequently after all three of these anesthetics that we cannot confirm or deny this statement. We are also unable to agree with their claim that anesthetization with divinyl ether is much smoother than with ether. On the contrary, the dogs given divinyl ether showed the most tendency to muscular excitement and not infrequently even mild clonic convulsions occurred during both the induction and recovery stages. The recovery stage, however, seemed to be somewhat shorter after the use of divinyl ether.

We were impressed by the superior smoothness of the analgesia produced by chloroform and it is easy to understand why the use of this drug has persisted in obstetrics, in spite of the growing appreciation of its greater toxicity. The smoothness of ether analgesia appeared to be intermediate.

The nutritional effects of a series of anesthesias averaging 32 for each dog are of some significance. In general, these repeated anesthesias seemed to be well tolerated so far as could be judged by the animal's appetite, appearance, and body weight. During the series the dogs receiving ether averaged a 3.1 per cent gain in weight, those receiving divinyl ether gained 2.3 per cent, and those receiving chloroform lost 1.3 per cent. Comparatively few respiratory infections were encountered. Whenever such infections occurred the animals were temporarily or permanently discarded.

## SUMMARY AND CONCLUSIONS

1. We have attempted to determine in dogs the margin of safety between the states of analgesia and respiratory arrest produced by ether, divinyl ether, and chloroform. Our aim has been to make our method conform, as far as possible, to the established principles governing biological assays. In our hands, chloroform had the highest potency but the lowest margin of safety. Ether, the least potent of the three, had the highest margin of safety. Divinyl ether with respect to both these characteristics occupied an intermediate position. Concerning the smoothness of the analgesia, we found that chloroform was the most satisfactory and divinyl ether the least.

2. A considerable variation was found in the response of our individual dogs to all of these drugs. Variation in the response to drugs is apparently inherent in all biological material and, for this reason, we believe that the inhalation method of anesthesia, because it permits greater individualization of dosage, is fundamentally safer than the method of predetermined dosage.

3. Although 121 toxic doses of chloroform were administered by a very brusque method, only one instance of primary chloroform collapse was encountered. We attribute this relative absence of primary circulatory collapse to the protective action of a high percentage of oxygen upon the heart.

4. A systematic study of the ease and certainty of resuscitation from respiratory arrest was carried out. Resuscitations from 45 overdoses of divinyl ether and from 42 overdoses of ether were uniformly successful, but following 49 overdoses of chloroform there were 6, or 12.2 per cent, failures to resuscitate. The high mortality of chloroform overdosage is attributed to the disastrous combination of a slow elimination and a high toxicity to the circulatory system which complicated the resuscitations.

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# THE SWOLLEN ATROPHIC HAND

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THE disease here reported combines swelling with atrophy in one hand. Both atrophy and swelling may vary in degree and, moreover, are not strictly correlated; wherefore the appearance may differ from case to case. Nevertheless, these two signs are always present, and their characteristic contrast is pathognomonic.

Fourteen patients were observed during the past 3 years. The swelling was stated to have appeared "over night" and to have persisted without change for several days or weeks past. Pain "resembling toothache" accompanied it. Two of the patients were referred to us for suspected fracture of the wrist, some slight injury being supposedly related to the symptoms. In 3 other instances, a fall from a height of several meters had preceded the development of swelling by 4, 7, and 8 months respectively. In the 9 remaining patients, there was no history of trauma.

The swelling involved the whole hand and wrist in 5 cases; only the wrist, in 2; and one or two fingers in 7 instances. It was resilient, giving the impression of being localized in some deeper layer beneath the epidermis, and it was not confined to the periarticular areas. There was no pitting on pressure. The swollen region did not differ in color from the normal skin. Together with the swelling, but sometimes masked by it, there was a variable degree of atrophy of the interosseous muscles of the affected hand, as evinced by increased concavity between the thumb and index finger and by flabbiness of the intervening tissues.

The skin of the fingers was thin and smooth, and in 2 instances slightly glossy. The ridges and furrows on the dorsal surfaces of the fingers, especially proximad to the nails, were either less numerous or less prominent than on the normal side; in 5 cases they were entirely absent. The nails were less shiny than on the healthy side, and the hairs were less numerous. The atrophic skin was neither abnormally dry nor abnormally moist, and neither warmer nor cooler than on the normal side. It could not be raised easily from the subjacent tissues; but when the swelling subsided, it appeared thinned and easily pliable. There were no discolored patches, no cracks, no scales, no prominent vessels, and no signs of impaired healing tendency.

The joints were freely movable; there were no signs of arthritis and no manifestations of rheumatic fever.

The reaction of degeneration was definitely present for the muscles of the arm on the affected side in 4 cases, and questionable in 1 case. Sensory disturbances were revealed by the history rather than by objective findings. All the patients had been troubled off and on, for periods extending from several weeks to 20 years past, by recurrent "rheumatic" pain in the shoulder girdle or in the deltoid region of the diseased side; by tingling sensations in the finger tips sometimes amounting to violent "electric shocks"; by the progressive inability to recognize and to hold between the finger tips some fine or thin object such as a needle or a sheet of paper. Objectively, however, there was neither increased nor diminished sensitivity to mechanical and thermic stimuli. In several cases, besides the interosseous muscles a group of muscles of the arm was found atrophic. Several patients had experienced definite weakness of the arm on the affected side; 2 of them stated that they often had difficulty in lifting a cup from the table. The grip was weak on the diseased side in all the patients.

In 6 instances, roentgen examination showed variable degrees of rarefaction of the bones of the affected hand; either diffuse, or confined to circumscribed areas in the metaphyses of the phalanges. Rarefaction was most marked in the carpal bones, where it amounted to almost complete demineralization in 2 instances. Unlike atrophic (proliferative, rheumatoid, infectious) arthritis, the joint spaces were normal in width even in the presence of very pronounced rarefaction.

From the Department of Roentgenology, American University of Beirut.

In 3 instances, the symptoms developed gradually, as already mentioned, within a number of months following a fall from a considerable height. Although these patients had not experienced any symptoms in the neck, an injury of the cervical spine was suggested clinically by the findings here described. Roentgenograms showed in 1 case traumatic destruction of two cervical intervertebral discs; in the second, fracture of one cervical articular process with forward displacement of the fragment; and in the third, fracture luxation of the third cervical vertebra with narrowing of the adjacent intervertebral spaces.

In 3 other instances, we were able to watch the gradual development of swelling and atrophy. These 3 patients underwent roentgen examination of the cervical spine because of recurrent pain and stiffness in the neck. In the first, the ridges of the right index finger became flattened 9 weeks after the onset of pain in the neck; swelling of the index and middle fingers occurred 2 weeks after this atrophy of the skin had become noticeable; the interosseous muscles became atrophic several months after the swelling had subsided. The second patient felt violent tingling in the right middle finger 7 months after pain in the neck had developed; swelling and atrophy of the skin developed simultaneously; but atrophy of the interosseous muscles was less definite than in the other patients of this series. The third patient had been treated for severe pain in the neck 11 months before the left hand had become weak and swollen. The swelling involved the whole hand; together with very marked atrophy of the skin of the fingers, this somewhat resembled scleroderma; but after the swelling subsided, the skin appeared very thin and loosely attached; there was no rigidity and no thickening of the areas involved.

In all the patients, roentgen examination revealed pronounced narrowing of one or two intervertebral foramina of upper cervical spine. This was confined to the diseased side; the corresponding foramina on other side were either normal in width or definitely wider than those on affected side. A simple device (Fig. 1) permits the foramina to be radiographed in identical projection on either side; variations due to different projection are thus excluded.

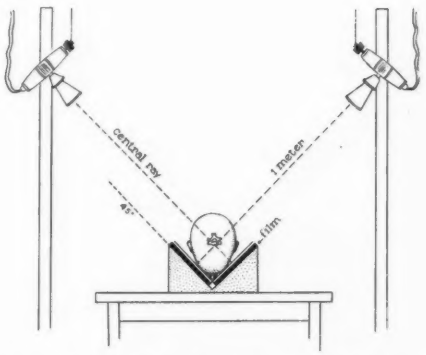


Fig. 1. Technique for roentgenographing the cervical intervertebral foramina in identical projection on both sides. The cervical intervertebral foramina are rotated forward, forming an angle of 45 degrees with the sagittal plane. Hence, in order to obtain correct optical sections, roentgenograms must be taken in oblique projection. The head of the patient rests upon a device which consists essentially of two tunnels standing at right angles, each forming an angle of 45 degrees with the horizontal plane. The midline of this device is exactly on the center of a roentgenographic table. Nose and chin of the patient, pointing vertically downward, are in touch with the junction of the tunnels. The tube stand is moved to the end of the table; right for the left foramina, left for the right. Tube stand at 60 centimeters from midline of table and device; tube 80 centimeters above table; anode-film distance 1 meter. These positions are identical for both exposures; merely the tube is rotated inward to an angle of 45 degrees, the central ray being at right angles with the plane of the film. The cervical spine is immobilized by means of immobilization of head and shoulders; deviations from the original position, even when slight, lead by unsymmetrical projection to differences in size and shape of the foramina: a common source of diagnostic errors.

The correlation of the clinical syndrome with the changes in the cervical spine, as found roentgenologically, is shown in accompanying Table I. The results would seem to indicate that:

1. Swelling and atrophy occur when the foramina above (and including) the fourth cervical are narrowed.

2. Swelling and atrophy of the hand are independent of the nature of the disease that produces this narrowing.

3. Rarefaction of the bones of the diseased hand occurs in the presence of swelling and atrophy of the skin, but is independent of atrophy of the interosseous muscles.

4. There may be no symptoms referred to the neck even in the presence of well marked bony changes of the cervical spine.

## TABLE I.—CLINICAL AND ROENTGENOLOGICAL FINDINGS

The width of the foramina is measured on the film; the figures indicate in millimeters the shortest transverse and vertical diameters. According to the laws of projection, the actual width is determined by the relation of the anode-film distance to the distance between foramina and film; but since the distance between foramina and film varies between 7 and 10 centimeters, the exact amount of distortion cannot be determined. This is insignificant, for, using the same technique on either side, the distortion is equal on both sides. In reality, the foramina are less than 10 per cent narrower than on the film; but it is merely the difference between the normal and the affected side which is of interest. The Roman numerals refer to the segments involved; e.g. C IV indicates the foramen between fourth and fifth cervical vertebræ, corresponding to the fourth cervical nerve root.

| Case and X-ray Nos. | Sex — Age — Race | History | Side diseased | Swelling | Skin | Muscles | Bones of hand (X-ray) | Cervical foramina | | | Type of disease underlying the constriction of foramina | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Nr | Diameters in mm. | | | |
| | | | | | | | | | Left | Right | | |
| 1<br>6689 | m<br>34<br>Armenian | Fall from several meters' height 5 months ago. Gradual development of pain and swelling in left hand. Left arm weak | L | Severe on wrist | Severe atrophy on finger tips | Interosseous and deltoid on left side atrophic | Rarefaction (Fig. 3) | C IV | 4 : 10 | 7 : 10 | Compression fracture with fracture of articular process | Reaction of degeneration strongly positive |
| 2<br>61252 | m<br>34<br>Arab | Fall from a considerable height 7 mo. ago. Gradual weakening of left arm. Severe tingling pain in left hand | L | Moderate on wrist | Severe on left fingers (Fig. 6a) | Deltoid and interosseous atrophic | Slight rarefaction | C III<br>C IV<br>C V | 1 : 6<br>2 : 9<br>2 : 9 | 5 : 9<br>5 : 10<br>8 : 9 | Fracture of articular process, traumatic thinning of intervertebral discs | Reaction of degeneration positive |
| 3<br>Pr 22 | m<br>22<br>Arab | Fall from over 5 meters' height about 9 mo. ago. Pain in shoulders, weakness of right hand | R | Marked of wrist | Severe of right fingers | R deltoid, biceps, and interosseous atrophic | Normal | C IV<br>C V | 8 : 9<br>9 : 9.5 | 3 : 9<br>7 : 9.3 | Compression fracture of cervical vertebræ | |
| 4<br>7482 | m<br>57<br>Arab | Rheumatic pain in shoulders for many years past. Severe pain in right arm for the past 3 months. Right hand became weak. Complete disability | R | Slight of wrist, more marked of fingers | Atrophy on fingers, subsiding under treatment | R interosseous, deltoid, and biceps, atrophic | Very slight rarefaction | C IV<br>C V | 6 : 11<br>7 : 11 | 5 : 9<br>3 : 9 | Exostoses, thorn shaped, at posterior borders of vertebral bodies (Fig. 2) | Reaction of degeneration positive |
| 5<br>Pr 23 | f<br>50<br>Armenian | Cancer of left breast, removed. X-ray therapy for local recurrency. Sudden swelling and pain in left hand | L | Marked of wrist and fingers | Atrophy nailsbristly slight glossiness (Fig. 6 b) | Left interosseous atrophic | Rarefied (Fig. 5) | C III | 2 : 9 | 9 : 9 | Rarefaction of left 4th superior articular process; with rounded exostosis bulging into foramen (Fig. 5) | |
| 6<br>6810 | m<br>47<br>Arab | Three years previous, awoke one morning to find his neck immovable. Pain in shoulder and left arm ever since. Left hand became sore and swollen | L | Marked of wrist (Fig. 6 c) | Atrophy | Left interosseous atrophic (masked by the swelling) | Severe rarefaction (Fig. 4) | C III<br>C IV | 4 : 10<br>4 : 12 | 7 : 10<br>5 : 12 | Posterior subluxation of vertebral body (Fig. 4) | |
| 7<br>6588 | f<br>59<br>Anglosax. | Chronic recurrent cystopyelitis. In Dec., 1936, severe pain and stiffness of neck. In Nov., 1937, swelling of left hand, with numbness and pain | L | On fingers (Fig. 6 d) | Atrophy on fingers | Left interosseous atrophic | Rarefied | C IV<br>C V | 3 : 10<br>3 : 9 | 6 : 12<br>5 : 11 | Thinning of all cervical intervertebral discs, with reactive exostoses at adjacent vertebral surfaces | |
| 8<br>71053 | f<br>42<br>Anglosax. | Chronic sore throat, recurrent bronchitis. Recurrent "rheumatism" of neck. Sudden swelling of right fingers, with violent tingling | R | On 3d finger | Slight atrophy | Very slight atrophy of R interosseous | Normal | C IV<br>C V | 7 : 10<br>4 : 12 | 3 : 9<br>3 : 12 | Thinning of intervertebral discs | |
| 9<br>6614 | f<br>56<br>Anglosax. | Chronic recurrent pyelitis. Acute pain in neck, shoulder, and occiput, followed by tingling in right fingers | R | On index | Slight atrophy | Definite atrophy of R interosseous | Normal | C III<br>C IV<br>C V | 6 : 10<br>6 : 8<br>4 : 8 | 6 : 10<br>3 : 9<br>4 : 9 | Thinning of intervertebral discs with exostoses at adjacent articular process and vertebral bodies | |
| 10<br>61286 | f<br>60<br>Anglosax. | Acute pain and swelling of left hand | L | On wrist | Slight atrophy | Atrophic interosseous, masked by swelling | Normal | C IV<br>C V | 7 : 8<br>5 : 6 | 9 : 11<br>6 : 6 | Thinning of lower cervical intervertebral discs | |
| 11<br>51435 | m<br>62<br>Anglosax. | "Rheumatism" in left hand and elbow | L | On wrist | Atrophy on finger tips | L interosseous atrophic | Normal | C III<br>C IV<br>C V | 5 : 10<br>6 : 9<br>7 : 9 | 9 : 12<br>7 : 12<br>7 : 10 | Thinning of intervertebral discs, exostoses at articular processes | |
| 12<br>52260 | f<br>52<br>Jewish | Mild diabetes. Recurrent eczema. Recurrent pain and stiffness in neck for more than 20 yrs. past. Violent tingling in right fingers | R | On 3d finger | Atrophy on finger tips | R interosseous atrophic | Normal | C IV | 7 : 9 | 3 : 9 | Isolated spondylarthritis of right 4th cervical apophyseal articulation; with calcification of ligamentum flavum, and concentr. calcification around 4th foramen | |
| 13<br>61507 | f<br>57<br>Jewish | Hypertension. Slight renal insufficiency. Pain in shoulder girdle, off and on, for 4 yrs. past. Recently pain in right hand | R | On wrist | Atrophy on fingers (Fig. 6 a) | R interosseous atrophic | Normal | C IV<br>C V<br>C VI | 5 : 9<br>4 : 9<br>3 : 6 | 3 : 9<br>1 : 8<br>2 : 4 | Marked thinning of intervertebral discs, with exostoses at vertebral bodies and articular processes | |
| 14<br>52208 | m<br>43<br>? | Pain in left shoulder, tingling in left hand | L | On finger | Atrophy on fingers | L interosseous and deltoid atrophic | Moderate rarefaction | C IV | 4 : 9 | 8 : 9 | Exostoses at tips of articular processes | Reaction of degeneration positive |

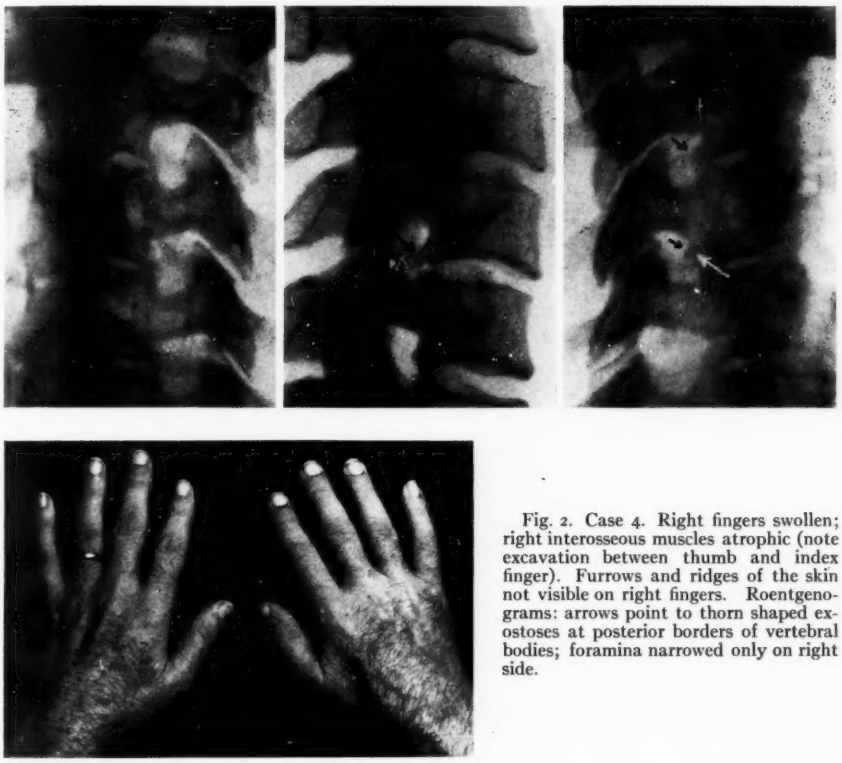

Fig. 2. Case 4. Right fingers swollen; right interosseous muscles atrophic (note excavation between thumb and index finger). Furrows and ridges of the skin not visible on right fingers. Roentgenograms: arrows point to thorn shaped exostoses at posterior borders of vertebral bodies; foramina narrowed only on right side.

Inactivity is not the sole cause of muscular and bony atrophy. Lesions of the cervical or lumbar vertebræ have caused atrophy of the muscles of an arm or leg, but the bones, despite disuse, appeared normal on roentgenograms more than a year after onset of disability. On the other hand, 5 years after acute "sciatica," severe and persistent atrophy of the muscles of one leg was noted. In complete absence of pain, the muscles did not regain normal strength and volume in spite of regular use.

### DIFFERENTIAL DIAGNOSIS

Swelling is not very common in neuritis. If present, it is described as being accompanied by vascular disturbances, by discoloration, and by hyperesthesia or analgesia of the skin, as, for example, in causalgia, glossy skin, and allied conditions. In our cases, on the contrary, none of these changes occurred.

From scleroderma, the condition differs by the complete absence of the characteristic thickening and retraction. Bony rarefaction, in scleroderma, is strictly confined to the terminal phalanges where the skin lesion is most pronounced; while in our patients, rarefaction was diffuse and not confined to the areas in which the skin was atrophic. With senile and idiopathic atrophy of the skin the condition has nothing in common; and from acrodermatitis atrophicans (Herxheimer), it differs by the absence of redness, of wrinkling, of prominence of veins, and of symmetrical or systemic involvement, and by the presence of subjective symptoms. From the atrophy of the skin and muscles, a condition that is sometimes associated with chronic atrophic arthritis, the lesion is easily distinguished by the entire absence of any arthritic manifestations in the hand.

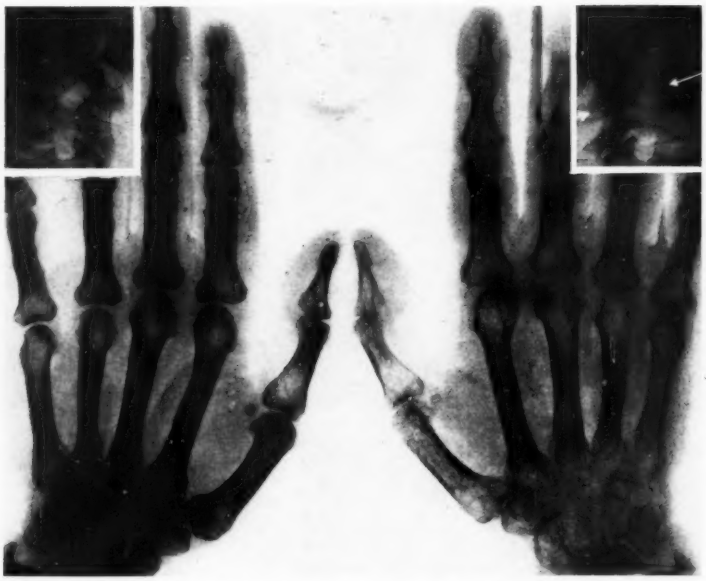

Fig. 3. Case 1. Bones of right hand demineralized. Right fourth foramen narrowed by displacement forward of a fractured fifth superior articular process (arrows). Left side normal.

It would seem that the changes under discussion are similar in nature to those trophic lesions which are occasionally observed in chronic diseases of the spine, as, e.g., "symptomatic" herpes zoster (18).

### THERAPY

Only 7 of the 14 patients underwent treatment. They were given a series of ultra-short wave treatments over the neck ("6 meter wave length"; electrodes at 10 centimeters distance from the skin on either side of the neck; each treatment 20 minutes). A total of from 5 to 24 treatments was given over periods varying from 1 to 10 weeks. Six of the patients responded almost immediately: pain and swelling subsided after the third or fourth treatment and did not recur during the subsequent 7 to 13 months (the patients are still under continuous observation). In the course of several months after termination of the therapy, both the muscles and bones regained normal or almost normal appearance, while slight degrees of atrophy of the skin persisted. Treatment was often prolonged because of pain in the deltoid region, arm, or neck.

In one instance (Case 1 of the table), no result was obtained either by this or by other modes of treatment; in this patient a fractured articular process was displaced into the intervertebral foramen. Operation was proposed, but not accepted.

No other therapy whatever was used. Since the aspect of the narrowed foramina did not change after the treatment, it is probable that this therapy acted upon the nerves, e.g. by decongestion of their sheaths.

### CORRELATION OF RADICULAR NEURITIS WITH BONY CHANGES AROUND FORAMINA

The findings seem to indicate that the syndrome here described is a trophic disorder caused by pressure upon cervical nerve roots within the intervertebral foramina. It has been known clinically for more than a century that changes in the limbs may be the result of chronic disease of the spinal column; but, up to the present, direct and conclusive proof of

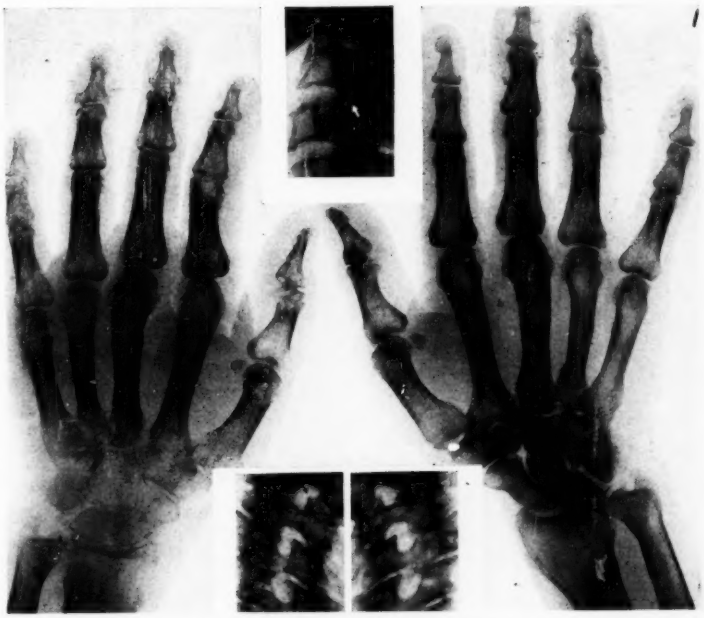

Fig. 4. Case 6. Bones of left hand greatly rarefied. Posterior subluxation of third vertebral body (no trauma). Step formation on both sides in the posterior margins of the fourth foramina, and in the left third foramen.

this pathogenetic mechanism is so difficult to obtain that the correlation has been frequently (and again very recently) denied or questioned (6, 15). It is true that spinal changes believed to cause radicular neuritis[1] are often found also in persons who are free from all symptoms. But this is due to the inconstancy of the relation between the width of the foramina and the caliber of the nerves that pass through them. The nerves are especially thick in the lower cervical and lower lumbar regions, where the foramina are not wider than in adjacent sections in which the nerves are more slender. Moreover, there are persons in whom the foramina are constitutionally wide, while in others, a certain amount of narrowing is physiological because of various postural factors. Hence it is the relative rather than the absolute amount of bony alteration which is responsible for compression of nerve roots; and it is natural that identical bony changes do not cause radicular neuritis in every instance. It is also true that neurologic symptoms identical with those found in certain chronic diseases of the column may occur in the absence of any demonstrable spinal changes. But this has little bearing upon the question here discussed; first, because radicular neuritis is obviously not pathognomonic of disease of the spine; and, second, because vertebral changes around the foramina do not become visible, as a rule, on "routine" roentgenograms. The statistics so far published are based upon anteroposterior and lateral views only; hence it is very probable that a certain number of spinal changes responsible for radicular neuritis have hitherto escaped recognition. Cases 4 and 5 show that these pathological formations appear to be insignificant in the conventional technique, although in reality they may reduce the width of the foramen to less than one-half of its original size (Figs. 2 and 5). Finally, it has been stated that on

[1]The term "radicular neuritis" is here used instead of "radiculitis," because the latter is a hybrid and because "radix," the root, is of ambiguous meaning. It would be more correct to speak of radicular nerve lesions, the inflammatory "-itis" not being verifiable. I am indebted to Dr. W. T. Van Dyck for his having suggested this modification.

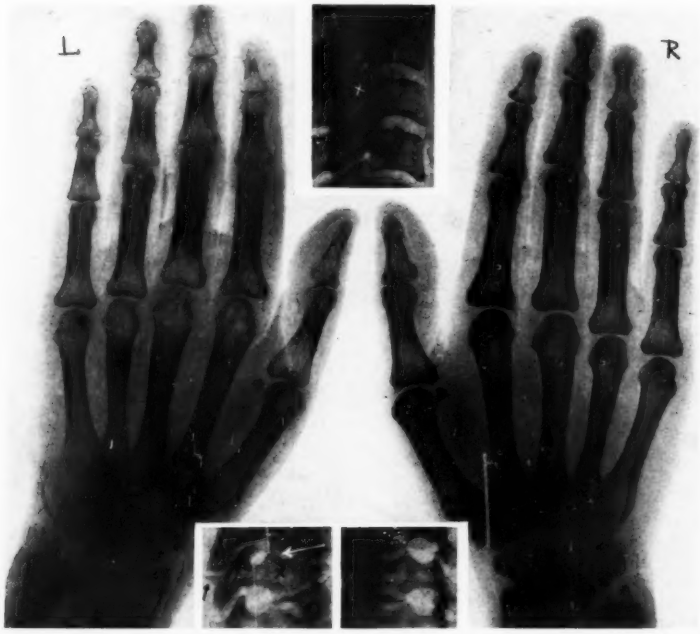


Fig. 5. Case 5. Bones of left hand rarefied. Left third articular process irregularly rarefied and thinned (cross); reactive exostoses at its posterior borders (black arrows). Rounded exostosis bulging into left third foramen (white arrow).

autopsy, bony changes around the foramina are not found to be sufficiently marked to account for compression of nerve roots. But the position of the articular processes—the main factor involved in narrowing of foramina—differs in autopsy from that found roentgenologically in the living patient because of the absence of postural load and muscle tone; moreover, dehydration of soft tissues postmortem may cause the nerves to appear thinner than *in vivo*, and slight alterations in the nerve, e.g. edema of its sheath, while causing well marked clinical symptoms, may not be verifiable on autopsy. Incidentally, no mention is made of histological examination of the nerves in those reports in which the correlation under discussion is denied.

It would seem, then, that the objections quoted are not significant enough to disprove the correlation of radicular neuritis with bony changes around the foramina. Considerable uncertainty, however, arises from the difficulties encountered in recognizing clinically both early and chronic stages of radicular neuritis. The classical syndrome of sensory and motor disturbances in segmental distribution is not always fully developed, and the symptoms are often indefinite or ambiguous (17). The patients may complain of vague rheumatism in the back and shoulders, radiating to the extremities; of moderate disability; of impairment of certain movements; of weakness of the grip; of paresthesia not confined to nerve segments. Objective sensory disturbances are very commonly not demonstrable; the reaction of degeneration is generally negative, and it is not until, in the course of many years, a group of muscles has become atrophic that radicular neuritis is suggested by these symptoms. As a rule, the correlation with a chronic lesion of the spine does not become evident, for the reason that the spinal section involved in a majority of cases is neither painful nor rigid.

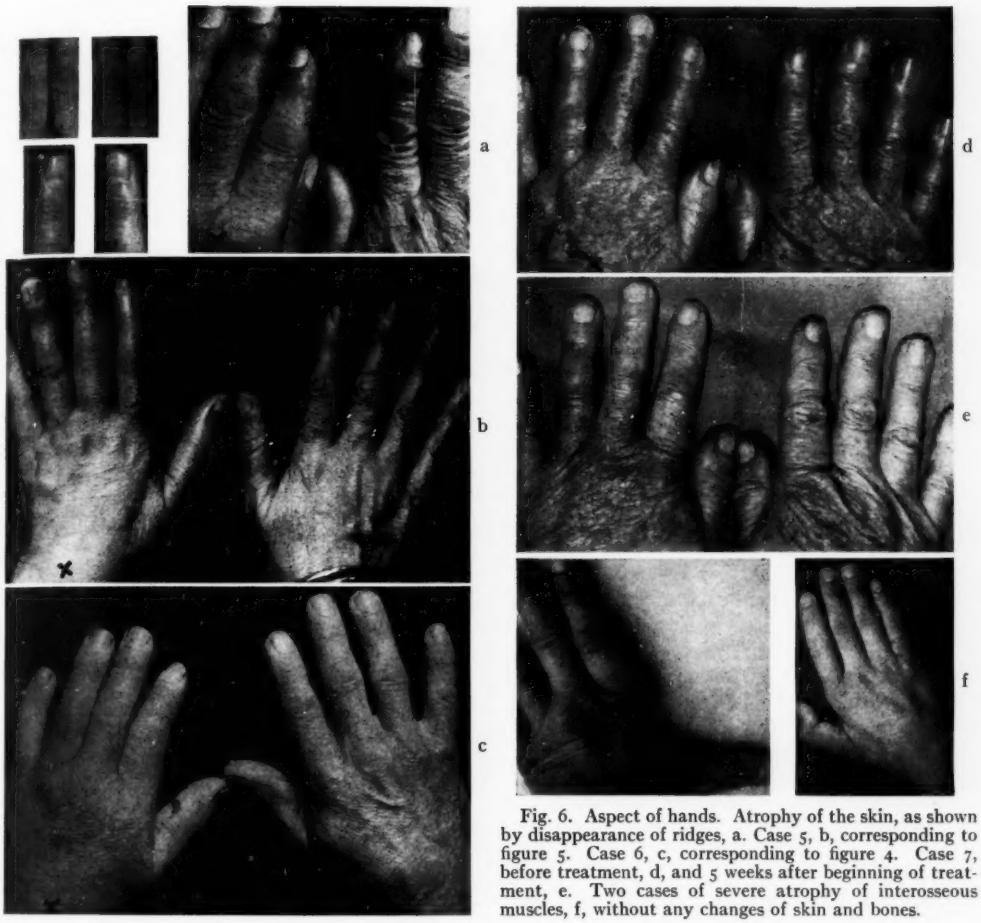


Fig. 6. Aspect of hands. Atrophy of the skin, as shown by disappearance of ridges, a. Case 5, b, corresponding to figure 5. Case 6, c, corresponding to figure 4. Case 7, before treatment, d, and 5 weeks after beginning of treatment, e. Two cases of severe atrophy of interosseous muscles, f, without any changes of skin and bones.

The various lesions that may produce narrowing of intervertebral foramina have been discussed in previous reports (12, 17). It may here be mentioned that the foramina are often *not* involved in the most conspicuous vertebral alterations—e.g. Pott's disease and hypertrophic spondylitis—whereas very slight changes in the intervertebral discs and articular processes often lead to marked narrowing of foramina. This is shown only on roentgenograms in which the foramina are projected in optical section; "routine" roentgenograms are here misleading, for they commonly fail to reveal the bony changes in question.

This report cannot in any way help to elucidate the question as to whether trophic disorders are due to lesions of hypothetical trophic nerves, or whether they are merely incidental to vascular, motor, and sensory disturbances (7, 11). But it would seem that the correlation of a particular type of trophic lesion with distinct bony changes around the corresponding intervertebral foramina is here demonstrated by the constancy of the spinal changes; by the almost experimental production in 3 cases of the trophic disorder as a result of injuries that caused constriction of the corresponding foramina; by the gradual develop-

ment, in 3 other cases, of the trophic disturbance following a period of severe pain in the neck due to bony changes in the cervical spine; by the occurrence, in cases of bilateral affection of foramina, of the trophic lesion on the side more severely affected; and by the results of treatment directed exclusively to the cervical spine.

SUMMARY

In 14 patients, a peculiar swelling accompanying atrophy of the skin, interosseous muscles, and—less constantly—of the bones of one hand was found to be correlated with unilateral bony constriction of intervertebral foramina in the upper cervical spine on the side of the affected hand. The clinical syndrome was independent of the nature of the disease that produced this constriction. In 6 of 7 patients treated, cure was obtained by ultra short wave therapy over the cervical spine. Atrophy of the bones was found to be correlated with atrophy of the skin, but was independent of atrophy of the muscles.

The development of well marked trophic lesions in an extremity affected, for many years, by "rheumatic" or "arthritic" pain, seems to indicate that in a certain group of cases, rheumatism may be due to radicular neuritis caused by chronic diseases in the spinal column.

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# NORMAL AND PATHOLOGICAL DEVELOPMENTS FROM THE CELLS LINING THE GRAAFIAN FOLLICLE

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THE object of this study is to follow the cells lining the primordial graafian follicle through their normal development and recession, and to trace the early beginnings of some of the pathological growths which can be shown to have their origins from these cells. Of the later developments of these growths much is known; but of their beginnings very little is known. Why they begin is a question the answer to which we have only an inkling.

The only structure in the cortex of the ovary aside from the dense connective tissue that makes it up is the primordial graafian follicle. The embryologists have taught us what happens to the ovum in the course of normal development; but the story of the lining cells of the graafian follicle is incomplete and often very hazy. To get a comprehensive view it is necessary to follow these cells through their many normal changes and to note the variations from the normal.

The primordial graafian follicle is very minute in size and contains a single cell, the immature ovum, and is lined by a single layer of small cells.

There is much confusion in the classification of the lining cells of the graafian follicle. They are commonly spoken of as epithelium or as germinal epithelium. This insistence on calling this layer of cells epithelium makes it practically impossible to explain their later development into connective tissue; because epithelial cells are considered a finished product, and when they reproduce, the new cells are the same as the original type.

On the other hand Ramon Cajal (1) states: "In 1918 Pedro Ramon successfully applying the methods of Golgi to the study of the graafian follicle has proved that the cells of the stratum granulosum as well as those of the discus proligerus are connective type." The conclusion of Pedro Ramon makes easy the following of these cells through their normal phases to the corpus albicans but makes difficult the explanation of the development from granulosa cells of several types of epithelium in the development of pathological growths.

Both of these difficulties are easily surmounted if we go back to the fact that these cells have the same embryological origin as the ovum; the ovum really is one of these cells that has been set apart. From the ovum is developed every type of cell found in the body. Since the cells lining the cavity of the graafian follicle are so closely related embryologically to the ovum, it is not surprising to see that they do not always follow a single line of growth, but may under varying stimuli produce a variety of types of cells.

The cells that are originally the lining of the primordial follicle are in the course of their normal development called by several different names. In the beginning they are called epithelium, or germinal epithelium or germinal cells. After the rupture of the follicle and the escape of the ovum, they are called lutein cells. When the lutein cells become hyalinized and form the corpus albicans, they are called connective tissue cells; in the atretic cysts they are known as granulosa cells. All these names are used for the same group of cells during normal development. In pathological growths it can be shown that the granulosa cells develop into several types of epithelium.

The normal graafian follicle follows one of two lines in its development:

1. The ovum grows in size, the lining cells increase in size and multiply rapidly, and finally the follicle ruptures and the ovum is extruded. The lining cells now called lutein cells form a mass of considerable size, the corpus luteum, which undergoes hyalinization and forms a connective tissue structure, the corpus albicans.

From the Department of Gynecological Pathology of the University of Maryland. Illustrations from the Art Department.

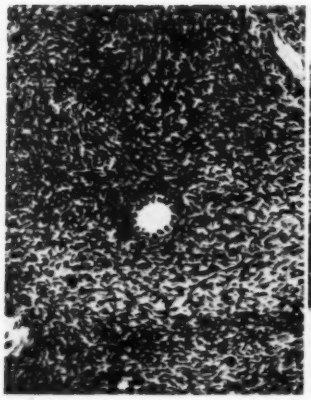

Fig. 1.

Fig. 2.

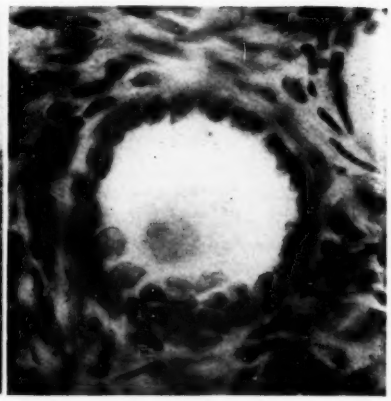

Fig. 3.

Fig. 1. Beginning development of the cells of a graafian follicle. A single layer of cells. The number of cells is increased and the size of the cells is increased.

Fig. 2. Three follicles are shown; one shows marked development; the other two are just beginning to develop.

Fig. 3. Higher magnification of the middle follicle shown in Figure 2. The lining cells are just beginning to proliferate inside the original layer.

2. King states: "The vast majority of graafian follicles present in an ovary do not become mature but undergo atresia. Just before the time of ovulation a number of follicles are approaching maturity. The rupture of one of these is accompanied by a regression of the others. This has been shown experimentally in birds by Pearl and Surface, and in rodents by Hermann and Stein, to be due to the presence of a hormone produced by the developing corpus luteum."

King further states that there "are various forms of regression: (1) absorption, (2) atresia with cystic development, (3) atresia with proliferation of the stratum granulosum and cystic formation, (4) atresia with proliferation of the stratum granulosum without cystic formation."

For present purposes interest centers on group 2, atresia with cystic development, and group 3, atresia with proliferation of the stratum granulosum and cystic formation.

Fig. 4.

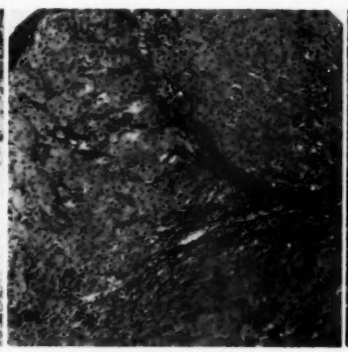

Fig. 5.

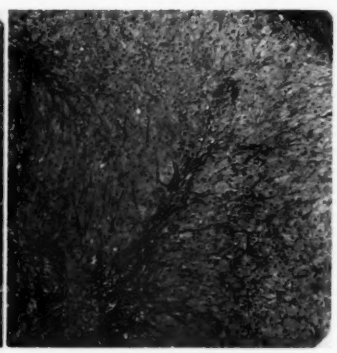

Fig. 6.

Fig. 4. An ovum with its nucleus and zona pellucida surrounded by layers of evenly distributed lining cells.

Fig. 5. A small portion of one lobule of an early corpus luteum. The cells are large; the outlines are difficult to reproduce; the nuclei are numerous and clearly defined. The lutean cells are definitely limited by the ovarian stroma.

Fig. 6. A corpus luteum a little older than that in Figure 5. The cells are paler and the nuclei are not quite so numerous.

Fig. 7.

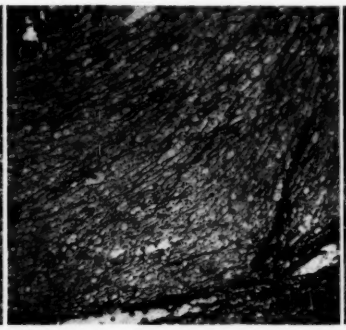

Fig. 8.

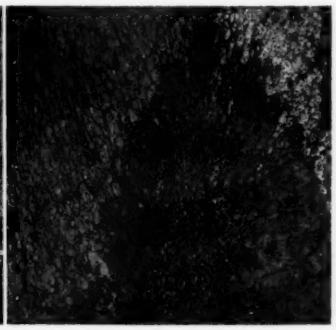

Fig. 9.

Fig. 7. A corpus luteum older than that in Figure 6 showing the progressive changes.

Fig. 8. Section showing still further degeneration of the cells.

Fig. 9. Almost complete hyalinization of the cells adjacent to the central degenerating blood clot. Outside of this area a group of cells that are rapidly losing their identity as lutean cells

NORMAL DEVELOPMENT OF FOLLICLES

When the graafian follicle begins to develop the first change observed is that the lining cells are increased in number and are increased in their size, but they form only a single layer (Fig. 1). As the growth continues, other cells appear inside the outer layer (Figs. 2 and 3), and these continue to form until the ovum is surrounded solidly by many layers of cells (Fig. 4). At this stage the ovum and the lining cells are much enlarged.

At some stage in the latter part of the growth of the follicular contents, a clear space over and outlining the discus proligerus appears, but in this series, no follicle showing this clear space has been found. The presumption that it is formed is founded upon the fact that every atretic cyst, which is only a graafian follicle that has been checked in its development, shows such a clear area.

Finally after the ovum and surrounding cells reach complete development and the cortex of the ovary has been softened by the new-growth of the contents of the graafian follicle, the ovum breaks through and is discharged into the abdominal cavity. The cells

Fig. 10.

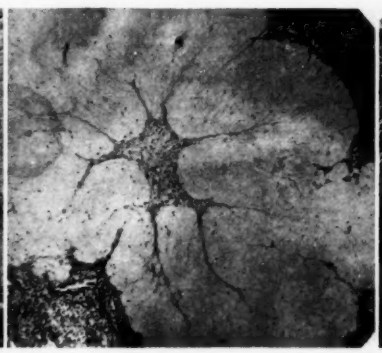

Fig. 11.

Fig. 12.

Fig. 10. The process of hyalinization is almost complete. A few nuclei are still present and there remain vague lines indicating old cell outlines.

Fig. 11. Hyalinization is practically complete. A few nuclei are still present. The whole mass had decreased in size to a small fraction of the original corpus luteum.

Fig. 12. A graafian follicle that has been checked in its development. The granulosa cells are clumped and drawn away from the theca. The outer layer of granulosa cells are very dark.

Fig. 13.

Fig. 13. A graafian follicle that has been checked in its development. A degenerating ovum. The granulosa cells are in contact with the theca. The outer layer of granulosa cells takes a very dark stain.

Fig. 14.

Fig. 14. The theca almost completely restored to the original type of connective tissue cells of the cortex.

Fig. 15.

Fig. 15. A relatively young atretic cyst. The ovum is well developed and shows the nucleus and zona pellucida. Surrounding it are the cells of the discus proligerus. The granulosa cells lining cavity are in many layers; outer layer stains more darkly than the others. A definite cleavage exists between the granulosa cells and the surrounding theca.

which remain in the ovary after the escape of the ovum are in the form of a mass called the corpus luteum. It is irregularly lobulated and has in its center an irregularly shaped space filled with blood. This space is relatively small but varies much in size.

The size of the corpus luteum, when compared with that of the primordial follicle from which it is derived, is an enormous body. The one from which this picture (Fig. 5) was made is about 12 millimeters by 18 millimeters. The field shown is only a portion of one lobule.

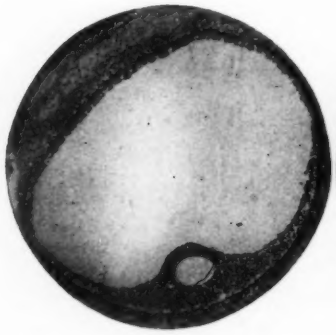

Fig. 16.

Fig. 16. An atretic cyst older than the one shown in Figure 15. More fluid has accumulated: the layer of granulosa cells is thinner. The ovum has a clear zona pellucida. The single layer of darkly stained cells marks the outer border of the granulosa cells; beyond these are the thecal cells.

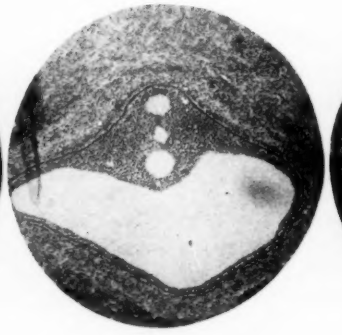

Fig. 17.

Fig. 17. An atretic cyst similar to Figure 16 except that in this one there are three rounded spaces in the discus. The one nearest the center of the cyst shows faintly the zona pellucida of an ovum. The outer space is very regular in shape but shows no nucleus and no zona pellucida. It is possible the graafian follicle, of which this atretic cyst is the remains, may have started to develop two or more ova.

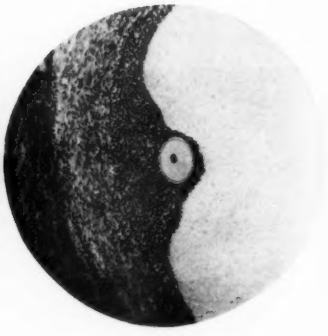

Fig. 18.

Fig. 18. Only a segment of a relatively large atretic cyst. The diminishing discus still holds an ovum with its nucleus and zona pellucida. The basal layer of dark granulosa cells marks clearly the limits of the theca on the side of the cyst and the dark cortex cells on the outer side.

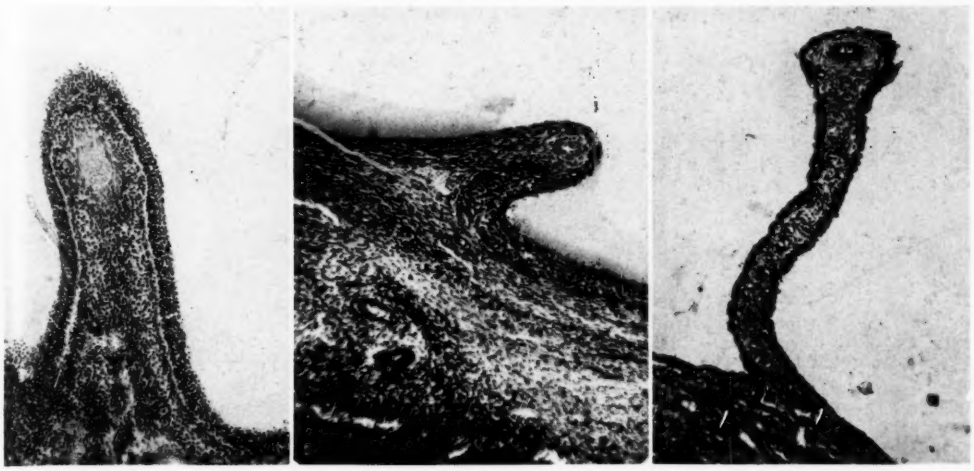

Fig. 19. Fig. 20. Fig. 21.

Fig. 19. An elongated discus in an old atretic cyst. It is covered by several layers of granulosa cells while the bulk of it is made up of immature connective tissue cells.

Fig. 20. A discus similar to Figure 18, but smaller. The granulosa cells are reduced to one or two layers, while the remainder of the mass of tissue is connective. Both have light areas near the apices which may indicate the location of the long degenerated ovum.

Fig. 21. A grotesque discus in a large atretic cyst, with small light spot near the apex present in Figures 18 and 19.

The bulk of the mass is made up of closely packed large cells with large dark staining nuclei. The line separating these cells from the ovarian stroma is sharply defined and this line of separation remains distinct throughout the process of recession.

### CORPUS ALBICANS

The formation of the corpus albicans from the corpus luteum is a recessional process. There is no new-growth of cells either lutein or cortical. The whole mass of lutein cells becomes smaller, the individual cells gradually lose color and diminish in size, and the nuclei become smaller and finally disappear. The process is very much like the hyalinization that takes place in uterine fibroids. The whole mass continues to decrease in size after the disappearance of the cell structure and ultimately disappears. This process of absorption is very slow and almost every ovary from an adult shows numerous corpora in various stages of disappearance (Figs. 6 to 11).

### THECA

When the graafian follicle begins to grow there is a change in the cells of the ovarian cortex immediately surrounding the follicle. These cells become edematous, enlarge in size, and take less hematoxylin stain than the unmodified cortex cells. This is the theca (Figs. 12 and 13). It is not a new-growth; it is not a part of the graafian follicle, and it takes no part in the formation of the corpus albicans. The nearest analogue to the cells of the theca are the decidual cells that form in the connective tissue of the endometrium or in the connective tissue of the fallopian tube when an ovum is developing in the immediate neighborhood.

This softening of the cortex is only a process of decreasing the resistance of the firm cortical tissue to allow the ovum to escape by rupture from the follicle into the abdominal cavity. The thecal cells persist especially around atretic cysts for a considerable time and gradually change back to their original type and merge with the other cortical cells (Fig. 14).

In the microscopical examination of ovarian sections, the identification of the theca is not absolutely necessary but helps in the identification of spaces that arise from graafian follicles. In the younger growths it is easily

Fig. 22.

Fig. 22. The remains of a discus in a large and old atretic cyst. A single layer of granulosa cells lines the cyst cavity on either side and extends over it. Everything beneath the layer is connective tissue.

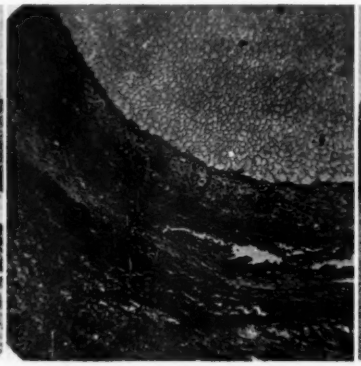

Fig. 23.

Fig. 24.

Figures 23 and 24 are from two sides of old atretic cyst to illustrate the variety in appearance of granulosa cells in the same cyst. Figure 23 shows a layer of granulosa cells of six to eight layers, Figure 24 only a single layer.

identified; but in the older ones in many instances the thecal cells have so nearly returned to their original status that they are difficult to distinguish from ordinary cortical cells. When present, they closely define the line between cortical and intrafollicular structures.

ATRETIC CYSTS

As has been stated previously when the mature graafian follicle ruptures, a hormone is thrown out by the corpus luteum that stops all the other developing follicles. It seems to have no effect upon follicles that have not begun to develop. One of the most common changes in the developing follicle that has been stopped in its growth is the formation of a small cyst, an atretic cyst. The lining cells are granulosa cells. They are much smaller than lutein cells and also smaller than those in partially developed follicles from which they were derived. The cells are piled up around the ovum and form the discus proligerus. Lining the remainder of the cyst, these cells are found in irregular layers of four

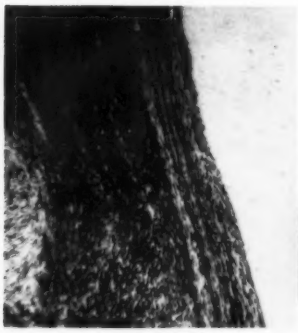

Fig. 25.

Fig. 25. The granulosa cells are reduced to a thin line. Beneath are the longitudinal connective tissue cells of the theca.

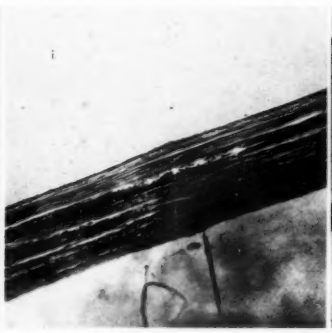

Fig. 26.

Fig. 26. A single thin layer of granulosa cells lining an old atretic cyst.

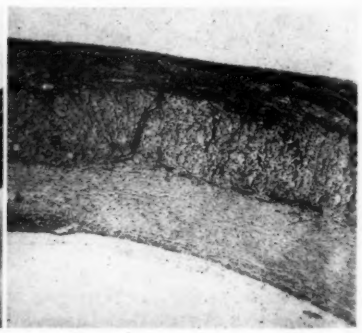

Fig. 27.

Fig. 27. A very low power picture of the whole thickness of the wall of a corpus luteum cyst. In the upper part of the picture is normal ovarian tissue. Just below that is a layer of lutein cells and below that newly formed connective tissue is seen.

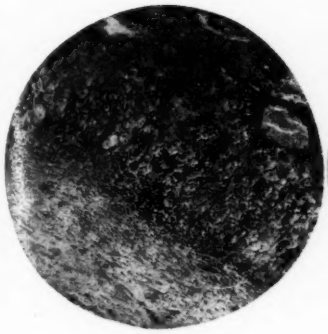

Fig. 28.

Fig. 28. The same section as shown in Figure 27 under higher magnification. A small segment of the ovarian cortex, the full thickness of the lutein cells, and a segment of the connective tissue which lined the cavity of the cyst are demonstrated.

Fig. 29.

Fig. 29. Goblet cells lining a small ovarian cyst. Intracystic pressure relatively low.

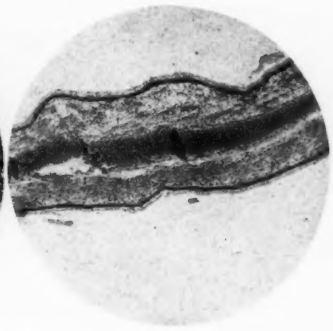

Fig. 30.

Fig. 30. Goblet cells lining two spaces in a multilocular cyst. Height of cells less than in Figure 28 because of greater intracystic pressure.

to eight cells deep in some cysts, while in others they thin out to one or two layers (Figs. 25 and 26).

The number of layers of granulosa cells in an individual cyst may vary greatly (Figs. 23 and 24). In some of the older cysts, the granulosa cells are evenly-thinned out to one or two compressed layers. A distinct demarcation is always present between the granulosa cells and the surrounding cortex of the ovary. Under normal conditions, there is never any intermingling of the granulosa cells and the thecal cells.

During the menstrual life of every woman, atretic cysts are found in the ovaries. They are normal products of the process of ovulation and consequently it is rarely necessary to remove them. Undoubtedly many of the larger thin-walled ones rupture spontaneously. Occasionally one is ruptured during a bimanual examination. Very rarely one attains such a size as to give rise to pelvic discomfort from pressure, or very exceptionally one has a twisted pedicle. On the whole the atretic cyst should be looked upon as a normal structure and left to take care of itself.

Fig. 31.

Fig. 31. A very low power magnification of an old atretic cyst. One side is lined by goblet cells which gradually fade into a single layer of granulosa cells. The other side has a single layer of granulosa cells (see Figs. 32 and 33).

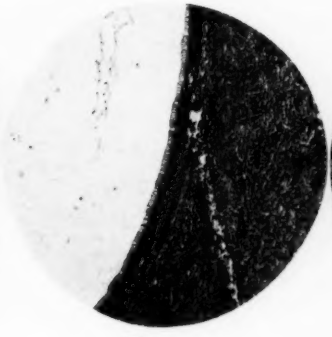

Fig. 32.

Fig. 32. A higher magnification of the lining on one side of the cyst shown in Figure 31. The goblet cells present are the same as are found in the adenocystoma.

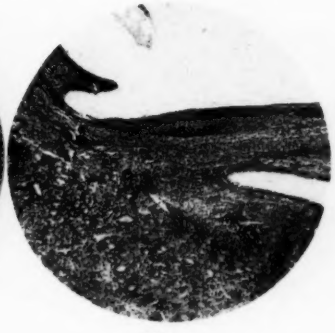

Fig. 33.

Fig. 33. Another portion of the lining of the cyst shown in Figure 31. There are no indications of goblet cells. Only a very thin layer of granulosa cells are present.

Fig. 34.

Fig. 35.

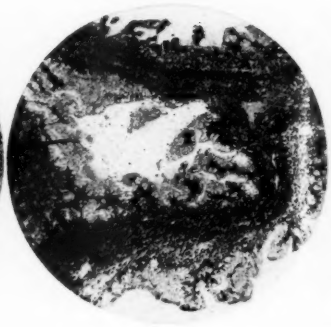

Fig. 36.

Fig. 34. A cavity lined partly by granulosa cells and partly by goblet cells.

Fig. 35. Gland-like spaces lined by goblet epithelium. In some parts there is a tendency to proliferate, suggesting a possible early malignancy.

Fig. 36. Small portion of an adenocarcinoma of ovary.

If a section of a comparatively recent atretic cyst is cut at a lucky angle, the discus proligerus will be cut through and the ovum will be bisected. The ovum is a large round clear cell surrounded by its capsule, the zona pellucida, and within is the nucleus and nucleolus. Heaped around the ovum is a mass of granulosa cells, the discus proligerus (Figs. 15, 16, and 17).

In the older atretic cysts the discus assumes a great variety of shapes, due to disintegration and change in type of the cells of which it is composed. These shapes vary from irregular heaps of cells to tongue-like projections, or small rounded firmly organized masses with no indication of the location of the ovum. Frequently the old discus consists of a mass of connective tissue with only a thin fringe of disintegrating granulosa cells covering it. Running straight across the field under the base can be traced the remains of the theca (Figs. 18, 19, 20, 21, and 22).

### LUTEIN CYSTS

The only apparent difference between lutein cysts and the much more frequently seen atretic cysts is the stage of development of the graafian follicles when their growth is stopped by the hormone thrown out from a mature corpus luteum. The lutein cyst is

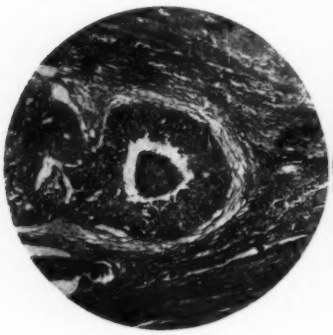

Fig. 37.

Fig. 38.

Fig. 39.

Fig. 37. In the center is a mass of detritus from an old atretic cyst. Where the lining of the cavity should be is a ringlike mass of carcinoma cells. Outside the space, the theca is clearly defined.

Fig. 38. An atretic cyst containing the remains of a discus and ovum. Projecting into it is a papillomatous growth.

Fig. 39. An old atretic cyst. The greater part of it is lined by granulosa cells. From one segment papillomatous tissue has displaced the granulosa cells and is growing into the cavity of the cyst.

Fig. 40.

Fig. 41.

Fig. 42.

Figs. 40 and 41. Both photomicrographs from the same atretic cyst. Figure 40 shows a beginning papilloma. Figure 41 shows no evidence of papillomatous tissue.

Fig. 42. Papilloma similar to that shown in Figure 39.

lined not by granulosa cells but by cells similar to those found in a corpus luteum. In the cyst illustrated is seen a thick layer of lutein cells next to cortical tissue. Between the lutein cells and the cyst cavity is a heavy layer of connective tissue cells (Figs. 27 and 28).

GOBLET CELLS

The granulosa cells in atretic cysts present a variety of appearances, and before it is possible to recognize pathological changes in them it is necessary to be familiar with the various normal types: the heaped up cells of the discus, the many layers in the newer cysts, and the thin pressed out cells of the older cysts. This last group is the most important, because it is from this group that the pathological cells develop (Figs. 25 and 26).

It seems hardly necessary to say that to learn the beginnings of pathological growths it is necessary to find them when they are very small and before they have caused much destruction or disintegration of the normal ovarian structure.

Fig. 43.

Fig. 44.

Fig. 45.

Fig. 43. A malignant papilloma. Section from another field on the same slide from which Figure 42 was made.

Fig. 44. An atretic cyst 11 by 7 millimeters. A small dermoid tumor, arrow, is growing within the cyst and is apparently at site of discus. Circa 5.6 diameters.

Fig. 45. A low power magnification showing the whole growth. Beneath the growth runs the remains of the theca showing that the growth is within the cyst. The light area of the growth is sebaceous gland material. The dark area facing the cyst cavity is made up of granulosa cells.

Fig. 46.

Fig. 47.

Fig. 48.

Fig. 46. One portion of the growth showing in more detail the granulosa cells and the sebaceous glands.

Fig. 47. A field showing one end of the growth with the granulosa cells piled upon the sebaceous gland growth and continuing beyond the growth in a single layer. Below the growth, and between the growth and the unmodified cells of the cortex, is a pale layer of cells—the remains of the theca.

Fig. 48. A dermoid growth showing sebaceous glands in the ovarian stroma and a growth of squamous epithelium that is creeping along the lining of an atretic cyst displacing the granulosa cells.

The goblet cell of the adenocystoma growing in a single layer with its dark base and white top is easily recognized by the amateur microscopist, because it is distinctive (Figs. 29 and 30). To find a group of small cysts in an ovary lined by goblet cells is no proof that they developed in atretic cysts. To find in one slide a group of small cysts, some lined completely with goblet cells and others lined completely by granulosa cells, is suggestive but only inferential evidence that the goblet cells developed from the granulosa cells; but when a small cyst is found partly lined by normal granulosa cells and partly lined by pathological goblet cells, it is practically conclusive that the goblet cells are developing from the granulosa cells. It is certain that the granulosa cells were there first.

Several slides showing both granulosa cells and goblet cells lining cyst walls have been found. Two of these are illustrated. One when the cyst was too large to allow sufficient magnification to show the character of the lining cells in one field is illustrated by three photomicrographs. Figure 31 shows the whole cyst with the goblet cells showing faintly on one side. Figure 32 is a higher magnification that brings out plainly the goblet cells; while

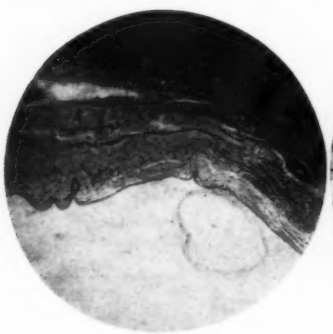

Fig. 49.

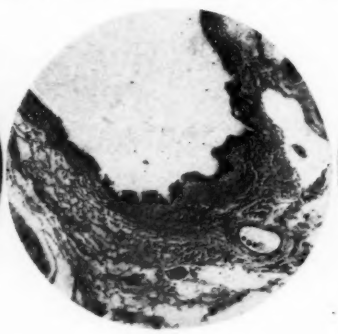

Fig. 50.

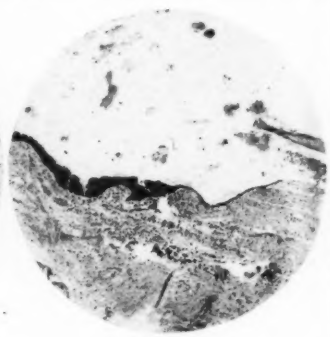

Fig. 51.

Fig. 49. Another portion of the wall of the cyst shown in Figure 48. Here the lining of the cyst wall is a single layer of granulosa cells.

Fig. 50. At one side, granulosa cells and the gradual transition to squamous epithelium are in evidence.

Fig. 51. Another field in the same cyst shown in Figure 50. The transition of granulosa cells to squamous epithelium.

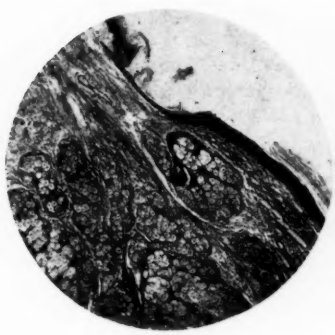

Fig. 52.

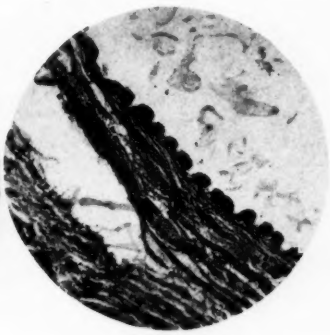

Fig. 53.

Fig. 52. Very similar to Figure 48. The replacement of granulosa cells by squamous epithelium.

Fig. 53. Another field of the same slide from which Figure 52 was made. This is a papilloma and is growing about 5 millimeters from the dermoid.

the opposite side of the cyst, Figure 33, shows only a thin layer of granulosa cells. In the second slide, the cavity was so small that both sides of it could be brought into one field and shows both the goblet cells and granulosa cells in one picture (Fig. 34).

The goblet cells are a type of true epithelium. From these cells carcinoma may develop. The fully developed carcinoma is usually very easy to recognize, but the one that is just beginning on its career of malignancy is open to various interpretations. Figure 35 illustrates such a condition. Various opinions have been expressed, and it is left as doubtful. On the other hand, Figure 36 is from a well developed ovarian adenocarcinoma.

Another type of carcinoma is shown in Figure 37. There is nothing to indicate the type of epithelium from which it developed, but there is fair evidence that it started in an atretic cyst. In the center is a mass of detritus such as is often seen in atretic cysts. Where the granulosa cells should be, is a thick circular mass of carcinomatous cells. Outside of the whole, a theca-like structure is clearly indicated.

### PAPILLOMA

The beginnings of papillomas of the ovary are very much the same as the development of the goblet cells of the adenocystomas. In numerous sections there can be shown the progressive development of the special type of epithelium which is found in papillomas from the granulosa cells of old atretic cysts. Figure 38 is clear evidence of a papilloma in an atretic cyst. We know the space is an atretic cyst by the presence of a portion of the discus containing a degenerated ovum. Projecting into the cyst is a papillomatous growth. This is no evidence that the growth started in this cyst. It could have broken through the wall of the cyst.

The beginning of a papilloma in an atretic cyst is shown in Figure 39. In this instance about three-fourths of the lining of a small cyst is granulosa cells. In the remaining fourth, a papilloma is developing directly from the granulosa cells.

In many instances, the early papilloma is found in old atretic cysts too large to be photographed as a whole. Figures 40 and 41 are made from such a cyst. In one is a dark thickened layer of cells that have begun to pile up. In the other a thin layer of cells that are only slightly modified granulosa cells.

All papillomas are potentially malignant but the degree of malignancy is very variable. Some have been known to grow for years with little invasion and very little destruction of tissue. Figure 42 shows no definite indications of malignancy while Figure 43 from the same slide is definitely malignant.

### DERMOIDS

According to Ewing it is generally accepted that dermoids of the ovary have their origins

in unfertilized ova. This assumption can be very nearly demonstrated in the specimen illustrated (Figs. 44, 45, 46, and 47).

The ovary from which this section was made was not enlarged and grossly showed a small atretic cyst. It was removed with a large uterine fibroid and the growth was discovered at routine pathological examination.

The atretic cyst measures 11 millimeters in its long diameter by 7 millimeters in the transverse. The growth occupies only a small segment of the circumference of the cyst. Outside of it the continuous line of the theca is clearly seen, demonstrating that the growth is intracystic. Piled upon the growth are many layers of granulosa cells, while the remainder of the cyst is lined by the ordinary thin layer of granulosa cells, commonly seen in old atretic cysts. The piled up granulosa cells indicate the position of the discus, and the position of the dermoid growth indicates that it started in the center of the discus, and that is where the ovum should have been.

In another section heavy squamous epithelium is seen creeping along the wall of an atretic cyst displacing the granulosa cells. Beneath the squamous cells are sebaceous glands and hair follicles. The opposite side of the same cyst is lined by granulosa cells not yet affected (Figs. 48 and 49).

In a third section, the heavy layer of squamous epithelium is seen lining a part of an atretic cyst and fading off into granulosa cells. On opposite side of same cyst squamous cells shift abruptly to granulosa cells (Figs. 50 and 51).

The first of these three sections shows sebaceous glands developing in a discus. The other two show the gradual change from granulosa cells to squamous epithelium.

One slide shows a cavity partially lined with growing squamous epithelium (Fig. 52), near it are sebaceous glands, hair follicles, and sweat glands. Within a few millimeters is a second cyst developing a papilloma, and also showing normal granulosa cells (Fig. 53).

In this series of pictures an effort has been made to show that the cells lining the primordial graafian follicle follow many lines of development. These variations in development are partially due to their embryological structure, and secondarily due to the influence of external agencies. We have some knowledge of some of these agencies. We know that the graafian follicle is induced to grow by the presence of a hormone. We know that when a mature graafian follicle ruptures it throws out a hormone that immediately counterbalances the growth hormone and stops the growth in all the other developing follicles.

The explanation of the further changes in the granulosa cells, which result from the first process, is a matter of speculation which in time will no doubt be cleared up. It seems reasonable to assume that since the normal changes are due to the action of hormones, the abnormal changes are due to a similar influence. As evidence of the correctness of this assumption, "William Cramer (2) and E. S. Horning have studied the carcinogenic properties of hormones. Estrogen is closely related chemically to some of the carcinogenic hydrocarbons. It is therefore not surprising that some of these induce estrus. But when tested by painting on the skin, the estrogens proved inactive. Subsequently, Lacassagne found that subcutaneous injection over many months produced mammary cancer, even in male mice. This has been confirmed by several workers. It therefore seemed that a substance can be carcinogenic for one organ (the mamma) and not for another (the skin). To test this point, Cramer and Horning applied a dilute solution of estrogen to the skin of male and female mice for a prolonged period. The surprising result was that cancer developed in the mamma but not in the skin, and as readily in males as in females. This result indicates an organ specificity for carcinogenic agents, a fact not previously suspected. Some apprehension has been aroused as to the danger of using estrogen, but Cramer and Horning have shown that carcinogenesis requires much larger doses and much longer periods than those of therapeutic administration."

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# SCOLIOSIS

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ALL students of scoliosis have appreciated two facts: (1) that early recognition of the deformity is the most important single factor in its management; and (2) that persistent and continuous treatment is necessary to obtain a satisfactory result. Early recognition of this lesion assures the opportunity of preventing the severe grades of the deformity, thus preventing the many and distressing physical, functional, and psychic complications. It is my intention to review the salient features of the symptomatology, etiology, and pathology of scoliosis so that we may have a clear concept of its clinical aspects, and to discuss the various forms of treatment, their application, and their results, in order that we may more accurately advise our patients.

## DEFINITION

Scoliosis is a deformity of the trunk in which the most conspicuous element is a rotary lateral curvature of the spine. Frequently we are inclined to think of scoliosis as relating only to the spine. This is an error. While changes in the conformation of the trunk and in the function of the viscera may be as difficult to recognize in the early cases as they are obvious in the advanced stages, yet pathological studies have shown that the thoracic cage and all of the thoracic and abdominal organs are involved in all instances and the change in morphology and function is in direct proportion to the severity of the deformity, of which the degree of the curvature of the spine is a fairly accurate index. Consequently, as a matter of convenience, scoliosis is described in terms of the curvature of the spine.

## TYPES OF SCOLIOSIS

There are two types of scoliosis, the functional and the structural. A functional scoliosis is a temporary physiological distortion of the trunk with a lateral deviation of the spine. This can be corrected by voluntary effort particularly after some instruction. There are no architectural changes in the skeleton and no alteration in the shape and function of the viscera. Functional scoliosis is common between the tenth and twentieth years. In a typical case of left dorsal scoliosis the spine is deviated to the left, the left shoulder is higher than the right, there is increased prominence of the left side of the back. The right iliac crest, commonly but incorrectly referred to as the "hip" is more prominent than the left. In a right dorsal functional curvature the findings are the opposite of those just described. When the patient bends forward the asymmetry of the back disappears completely. After some instruction the patient can stand erect with his back entirely symmetrical. An x-ray film made in the usual or supine position shows a normal spine and normal ribs.

## TREATMENT OF FUNCTIONAL SCOLIOSIS

A cure may be effected by the following procedure. All potentially causative agents should be removed. Thus a child should not be permitted to carry a heavy load of books continuously on one side of the body. The clothing should be adjusted so that there is no unequal pull on the shoulders. Visual and auditory deficiencies must be removed or compensated. Obesity must be rectified by dietary restriction. An explanation should be given to the patient, if old enough, of the nature of the faulty attitude and he must be patiently shown how to sit, stand, and walk correctly. Corrective and symmetrical gymnastic exercises should be taught and these should be performed daily for many months under the direct supervision of the physician or one specially trained in this type of work. These exercises improve muscle tone, serve as a stimulus to physical activity, and de-

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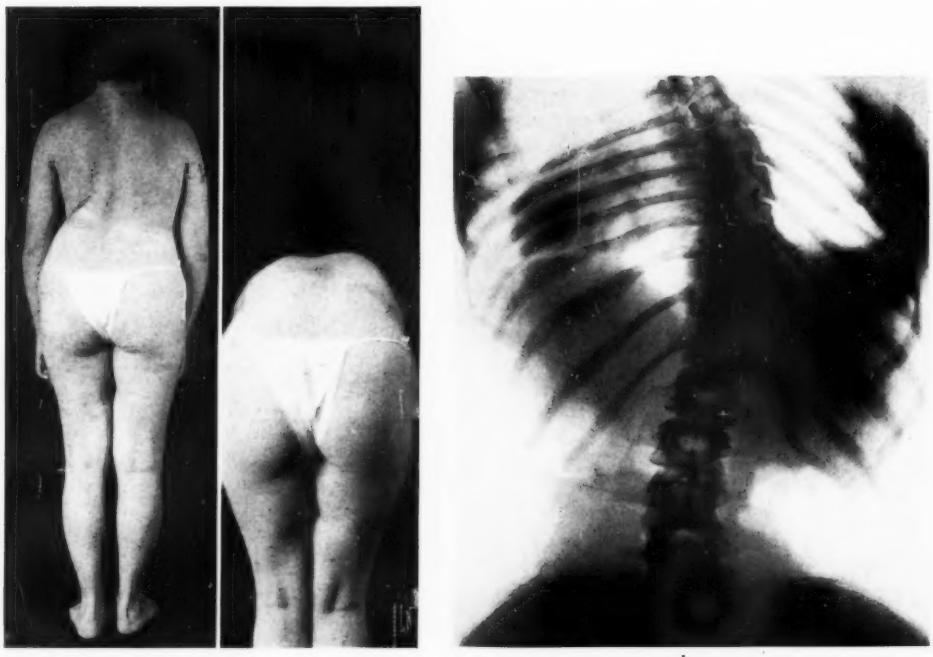


a b

Fig. 1. Right dorsal curve. a, Photographs of patient standing erect and bending forward. Note asymmetry of back, deviation of dorsal segment of spine to the right, prominence of left iliac crest ("hip"), incurvation of left side of abdomen and backward projection of ribs on right side. b, X-ray picture showing deviation of dorsal vertebræ to the right with apex of curve at seventh dorsal. Note increased angulation and separation of right ribs and approximation and reduced angulation of left ribs. Distance between spine and periphery of costal cage reduced on right side. Slight compensatory curve to left in lumbar region.

velop the habit of holding the body in a symmetrical attitude. The program of treatment must be carried out over 1 to 2 years to assure the permanency of the cure.

### STRUCTURAL SCOLIOSIS

Structural scoliosis though less frequent than the functional type is vastly more important because it signifies the existence of an organic change in the various tissues and not uncommonly grave functional visceral disturbances and psychic imbalance. A structural scoliosis is one in which there is a fixed deformity of the trunk which the patient cannot voluntarily correct. While all of the components of the trunk are involved in this deformity it is described in terms of the abnormality of the spine, the changes of which are the most apparent.

Structural scoliosis has been divided into three grades, mild, moderate, and severe, according to the degree of deviation and rotation of the vertebræ. The terms employed to describe the scoliosis relate also to the section of the spinal column involved, the direction of the convexity of the curve, and the known or likely etiology. For instance, one speaks of a moderate paralytic right dorsolumbar curvature, meaning that all or most of the dorsal and lumbar vertebræ are deviated to the right, the curvature is of a moderate degree, and has followed an attack of infantile paralysis.

A description of typical examples of structural scoliosis follows.

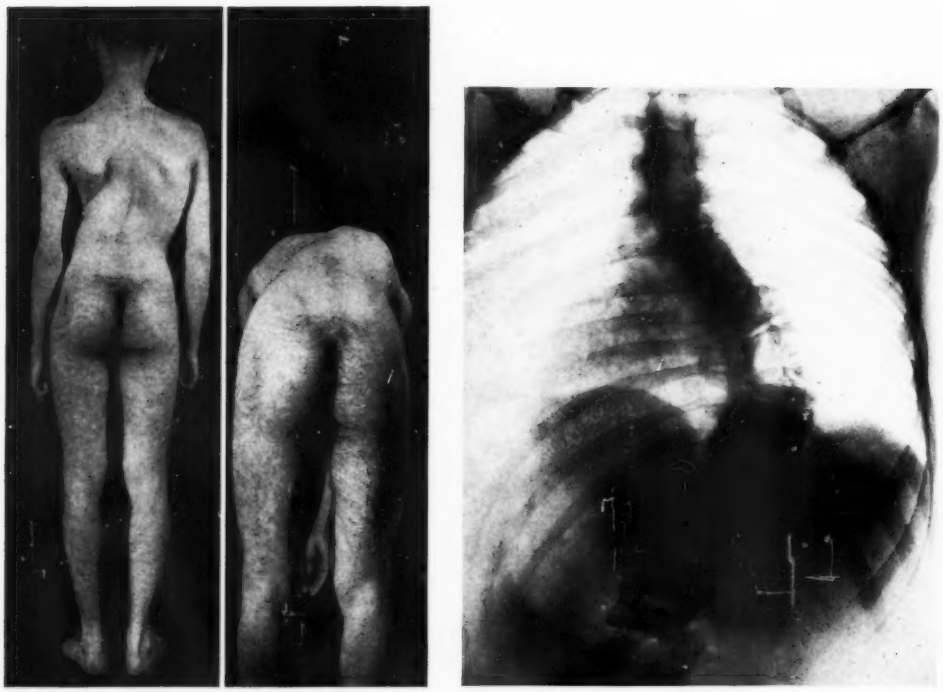
*Right dorsal scoliosis* (*Fig. 1, a*). In a posterior view of a patient with this deformity one observes that the right shoulder is higher

than the left. The ribs on the right side project backward. The right scapula is farther away from the midline and is more prominent than the left; its inferior angle projects backward conspicuously. There is a flatness or hollowness of the chest on the left side. The line of the dorsal spinous processes is deviated to the right. There is prominence of the left iliac crest, popularly called the left hip. This prominence may be further emphasized by deflection of the trunk to the right. The lumbar area may be symmetrical but more often the left side is slightly more prominent than the right, due to compensatory deviation and rotation of the lumbar vertebræ to the left. The neck may be symmetrical or the cervical vertebræ may be mildly deviated to the left. In some instances, especially in the severe grades, there may be an increase in the normal backward curve of the dorsal segment causing a kyphoscoliosis. The pelvic and shoulder girdles are usually in the same vertical plane. In some instances in which there is a marked lordosis or exaggeration of the normal lumbar forward curve, the shoulders may be in a plane considerably behind that of the pelvis. In the normal subject a vertical line dropped from the occipital protruberance will pass through the intergluteal fold. In a right dorsal scoliosis the cervical and lumbar vertebræ may remain in the same vertical line or the trunk may be tilted to the right so that the line of the occipital protruberance is 1 to 3 inches to the right of the intergluteal fold. When the patient bends forward the asymmetry of the back and the backward projection of the right ribs becomes exaggerated. Deviation of the spine is best noted in the erect position but rotation of the vertebræ, as evidenced by the backward projection of the ribs on one side, is seen to advantage when the patient bends forward. This fact is important because in instances in which there is only a mild deformity it may not be apparent in the erect position, especially in stout children, but is always and easily recognizable when the individual bends forward. The anterior view of the body will show an opposite deformity of the chest; the right side is flat and the left side prominent. This deformity

Fig. 2. Left lumbar curve. Lumbar vertebræ are deviated and rotated to the left; apex at midlumbar area. There is a slight compensatory curve in the dorsal segment, but the chest as a whole is altered but very little.

in a well developed girl in the adolescent period results in unusual prominence of the left breast. Frequently a girl is first brought to us for examination because of a "high hip" or a "big breast."

An anteroposterior x-ray film (Fig. 1, b) shows the dorsal segment to be deviated to the right with the apex of the curve about the mid-dorsal vertebræ. The dorsal vertebræ are not only deviated but rotated to the right. It is the rotation of the vertebræ that causes the angulation and backward projection of the ribs. The ribs on the right side have an increased inclination. The ribs on the left side are nearer to one another and flatter and their inclination is markedly reduced. The distance between the spine and the left side of the chest wall is greatly increased. The cervical and lumbar vertebræ



a b

Fig. 3. Right dorsolumbar curve. a and b, Apex of curve at tenth dorsal. Marked incurvation of left side of chest and abdomen. Note asymmetry of back and backward projection of right ribs.

may be in the midline so that the spine looks like a question mark. Usually there is a slight compensatory deviation to the left in both the cervical and lumbar vertebræ. At the apex of the dorsal curve, the vertebræ are wedge shaped, the degree depending on the severity of the curve.

The vertical diameter of the trunk is always reduced. In the mild curve this may be of no practical significance. In the severe type the reduction may be very noticeable with conspicuous shortening of the patient's height and telescoping of the chest into the abdomen so that the lower ribs may be very near to, in contact with, or even below, the iliac crests.

The thoracic and abdominal organs are involved to a degree commensurate with the severity of the curvature. In the mild cases the lungs, heart, and abdominal organs are so slightly altered as to give no evidence of it clinically. In the severe grades, however, there is marked distortion of the lungs, the heart is displaced, the abdominal viscera are crowded downward toward the pelvis. In this group of cases there may be not only pain in the back but dyspnea on slight exertion and a marked reduction in the capacity for physical exercise.

*Left lumbar scoliosis.* In a posterior view the most noticeable features are prominence of the left loin with hollowness and incurvation of the right loin. The spine presents a deviation of the lumbar vertebræ to the left with a variable degree of compensatory curve in the dorsal region to the right. The shoulder and pelvic girdles are in the same vertical plane. On palpation there is an abnormal softness in the right loin whereas in the left loin there is an unusual firmness, the fingers

feeling a bony resistance. In an anterior view the most notable feature is an incurvation of the right side of the abdomen with prominence of the right iliac crest.

An anteroposterior x-ray picture (Fig. 2) shows a deviation and rotation of the lumbar vertebræ to the left. In the midlumbar region there is wedging of the vertebræ. The extent of the deformity of the vertebræ depends upon the degree of the curvature. There is always some slight deviation of the sacrum to the right. In the severe cases there may be marked distortion of the pelvis, which in the female may be a menace during childbirth.

In a simple lumbar curve the thoracic organs are practically unaffected. The abdominal organs are only slightly affected, since most of them are in front of, and not in intimate contact with, the spine. The liver, spleen, and kidneys placed high up in the abdomen lie over the transitional vertebræ between the thoracic and lumbar segments, in which region there is a minimal deformity. Consequently, they are not affected except in the very extreme cases in which there is marked reduction of the vertical diameter of the abdomen.

*Right dorsolumbar curvature* (*Fig. 3, a*). When all of the dorsal and lumbar vertebræ are involved in a single curve the apex of the curve is usually at about the tenth dorsal vertebra, the curve beginning at the third or fourth dorsal and terminating at the lumbosacral junction. These curves vary from exceedingly mild in the so called idiopathic variety to extremely severe as is often seen in paralytic deformities. The posterior view will show an exaggeration of the findings of a simple dorsal curve. In the dorsolumbar curve there is often a marked deviation of the trunk to the right. The vertical diameter of the trunk may be reduced so that the right ribs are in contact with the right iliac crest and the left ribs below the left iliac crest, resulting in friction between these parts and pain in the back or abdomen. In an anterior view there is asymmetry of the chest and abdomen. The x-ray picture (Fig. 3, b) shows a single curve extending from the upper dorsal region to the lumbosacral junction with very mild involvement of the cervical region and rarely a distortion of the sacrum, except in the paralytic cases in which there may be a unilateral contraction of the pelvis or a very severe deformity of the entire pelvis.

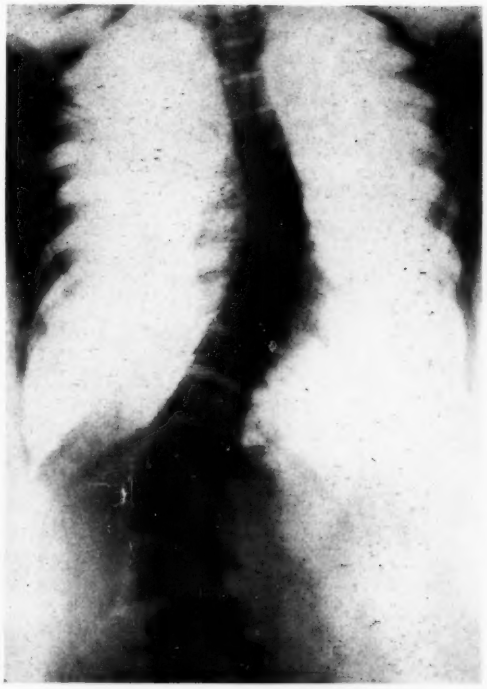


Fig. 4. Compound curve; right dorsal left lumbar. The dorsal and lumbar segments are approximately of equal extent and severity. The deviation and rotation elements in each segment are of equal degree.

*Right dorsal left lumbar scoliosis* (*Fig. 4*). This is the most frequent of the compound curves. The features of this curve combine those of the simple dorsal and the simple lumbar curves. In the typical right dorsal left lumbar curve the shoulders are usually on the same horizontal plane. The dorsal vertebræ are deviated and rotated to the right. The ribs on the right side project backward and are abnormally angulated. There is lateral compression of the right side of the chest and flatness or hollowness of the left side. There is prominence of the left loin and

Fig. 5. Congenital scoliosis. Note hemivertebra on left side between tenth and eleventh dorsal vertebræ and on right side between eleventh and twelfth dorsal.

TABLE I.—ANALYSIS OF 221 PRIVATE CASES OF STRUCTURAL SCOLIOSIS

| | Cases | Per cent |
|---|---|---|
| Type of curvature recorded in | 215 | |
| Right dorsal | 45 | 21 |
| Left dorsal | 5 | |
| Right lumbar | 8 | |
| Left lumbar | 33 | 15 |
| Right dorsal, left lumbar | 55 | 25 |
| Left dorsal, right lumbar | 4 | |
| Right dorso-lumbar | 42 | 19½ |
| Left dorso-lumbar | 23 | 10 |
| Degree of curvature recorded in | 119 | |
| Mild | 60 | 30 |
| Moderate | 87 | 44 |
| Severe | 52 | 26 |

flatness or hollowness of the right loin, with prominence of the right iliac crest. When the patient bends forward the asymmetry of the back becomes very evident. This type of curvature varies from the very mild to the severe as do all other curvatures. In an anterior view there is asymmetry of the chest and abdomen. The right side of the chest is flat and the left is convex. In the very severe types of this deformity there may be a telescoping of the chest into the abdomen with diminution of their capacity and functional disturbance of both the intra-thoracic and the abdominal organs (Table I).

### ETIOLOGY

There are two types of structural scoliosis, congenital and acquired. In the congenital group there are two subdivisions, one in which there is a recognizable deformity of one or more of the vertebræ or ribs, and the other in which there is no recognizable bony defect.

*Congenital deformity with manifest osseous defect* (*Fig. 5*). The commonest type is one in which there is an extra centrum or body of a vertebra. This is usually in the form of a wedge that is interposed between two vertebræ, causing a lateral deviation of the spine. The next is a wedge formation of a vertebra. The third type is a fusion of part of one vertebra with another, forming a wedge mass. The fourth is a spina bifida occulta of varying degree involving one or many vertebræ. All of these represent developmental anomalies or defects. They may be present singly or in any combination, and involve only one segment of the spine, several segments, or even the entire spine. In the majority of instances the curvature resulting from a congenital scoliosis with a manifest osseous defect is a simple curve and does not advance to an extreme deformation of the entire trunk. When there are multiple defects a severe kyphoscoliosis may result.

*Congenital scoliosis without manifest bony deformity.* This type is usually seen in early childhood or during infancy. The x-ray pictures show a scoliosis, but no abnormal formation of any of the vertebræ. It may be admitted that because of the slight ossification at this early age wedge vertebræ may not be sufficiently clearly outlined to be recognizable. But in many of these cases, observed over a period of years, it becomes apparent that the primary lesion was a maldevelopment and unilateral contraction of

the soft tissues, just as it is, for instance, in torticollis. In a study of 46 cases of congenital scoliosis in infants Harrenstein came to the same conclusion.

*Acquired scoliosis.* There are two groups, a small one in which the cause is recognized and a large one in which there is no discoverable causative factor.

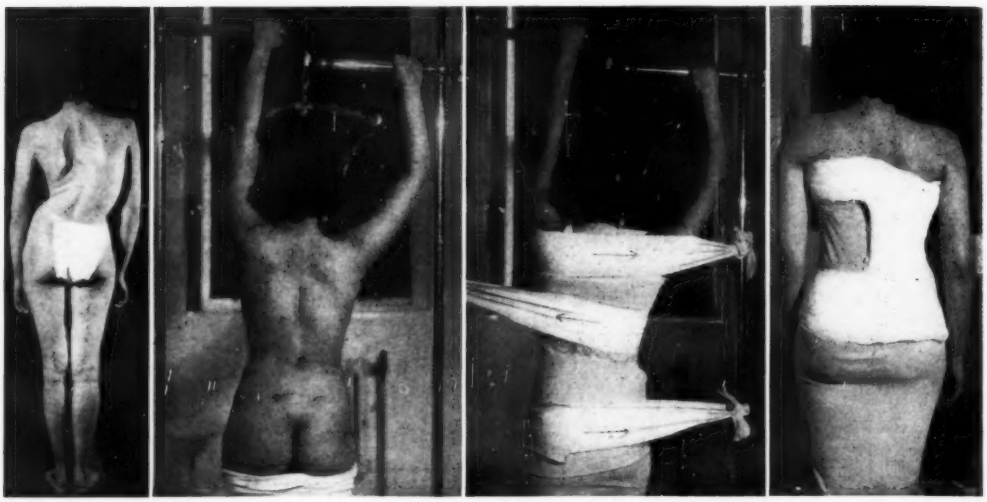
*Acquired scoliosis of known cause.* In the known forms there are scolioses that may be definitely attributed to the following reasonably certain causative agents:

*Rickets.* Rickets by disturbing the nutrition and the growth and shape of the vertebræ may cause scoliosis. Rachitic scoliosis is often severe and resistant to correction. In continental countries, where rickets is more common than in our own, it is said that rickets accounts for about 30 per cent of the cases of scoliosis. In this country, in which there has been a sharp decline in the incidence of rickets, there is only a comparatively small percentage of scoliosis in which rickets can be assumed to be the cause.

*Infantile paralysis* (*Fig. 6*). This is a comparatively frequent cause of scoliosis. In 1926, reviewing a large number of paralytic cases, I estimated that about 5 per cent developed scoliosis. This was confirmed in a recent review of 221 private cases in which there were 12, or 5 per cent, of paralytic origin. The cause of the scoliosis in this disease is muscle imbalance. It is not uncommon to see, for instance, a right dorsolumbar scoliosis in a paralytic child with paralysis or paresis of the muscles of the right side of the abdomen. Similarly paresis of one leaf of the diaphragm or of some spinoscapular or erector spinæ muscles will result in a spinal curvature.

*Lesions of the chest.* Empyema is a well known cause of scoliosis. The convexity of the curvature is away from the side of the empyema. The curvature is initiated by the persistence of the attitude of deviation of the trunk assumed to relieve discomfort. A single curve is formed with the convexity toward the well side. In a long drawn out illness the deformity may become severe. Recently there has appeared a type of scoliosis which is apparently secondary to the extensive thoracoplasties being performed for pulmonary tuberculosis. The etiology is dependent upon the loss of the bony wall of the chest by removal of some ribs. The scoliosis is always toward the side of operation. The deformity in the adult is mild. In children and in an occasional adult the deformity may be severe. It differs from the ordinary scoliosis in that there is a deviation but hardly any rotation of the vertebræ. Thus in the usual thoracoplasty scoliosis there is lacking the customary backward projection of the chest on the convex side of the curve. In those thoracoplasties in which large fragments of the ribs adjacent to the spine are left, a scoliosis does not develop. Perhaps in the future a satisfactory thoracoplasty will be possible leaving substantial segments of ribs near the vertebræ. To prevent the occurrence of scoliosis following thoracoplasty the operation of resection of the ribs has been combined with a spine fusion.

Fig. 6. Severe paralytic scoliosis. Note the marked deflection of the trunk to the right and the severe angulation and backward projection of the right ribs. The extensive muscular paresis accounts for the laxity of the tissues of the trunk and lack of voluntary control of posture. This type of scoliosis requires a spine fusion for stabilization of the vertebral column.



a b c d

Fig. 7. Reduction of curvature by forcible correction and application of plaster-of-Paris jacket. a, Photograph of patient with paralytic scoliosis before treatment was instituted. b, Patient suspended by Sayre halter. Note effect of longitudinal traction; this is more marked in paralytic than in idiopathic cases. c, Application of corrective forces by lateral traction straps. d, Plaster-of-Paris jacket maintaining patient in corrected attitude.

*Inequality of the lower limbs.* A slight disparity in the lower limbs will not cause a scoliosis. When, however, because of disease in or deformity of a lower limb there is a marked inequality, with a shortening of an inch or more, there is a deviation of the lumbar spine to the side of the shortened limb, with a compensatory curve in the dorsal region in the opposite direction. It does not necessarily follow that when one leg is shorter than the other a scoliosis will ensue, but an inequality of the lower limbs should be compensated during adolescence at least by an appropriate lift in the corresponding shoe.

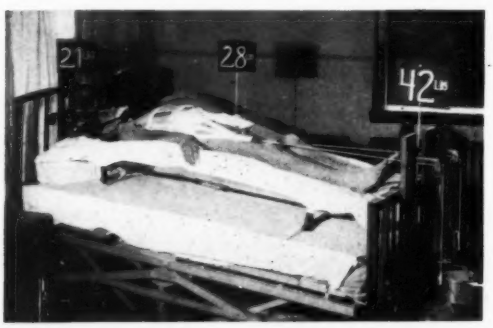



Fig. 8. Correction of curvature by traction on a convex frame. Traction obtained through weights. As much as 125 pounds have been used with very little discomfort to the patient. The blow bottles are used several times a day for thorough aeration of lungs.

*Defective vision* or hearing may cause pos tures which may initiate a scoliosis.

*Torticollis*, particularly the so called congenital variety, is accompanied by a secondary scoliosis.

*Neurological disturbances* such as syringomyelia, Friedreich's ataxia, and spastic paralysis have frequently an associated scoliosis.

*Heart disease* does not generally cause scoliosis, but in the exceptional case in which there has been an enormous hypertrophy of the heart a scoliosis is seen. It is difficult to be certain of the exact mechanism of the pathogenesis of the scoliosis. The important point in this relationship is to appreciate the imperative need to treat the heart condition, to minimize its ill effects, as that is the best means of controlling the scoliosis.

*Hereditary scoliosis.* In certain families there is a distinct tendency to scoliotic de-

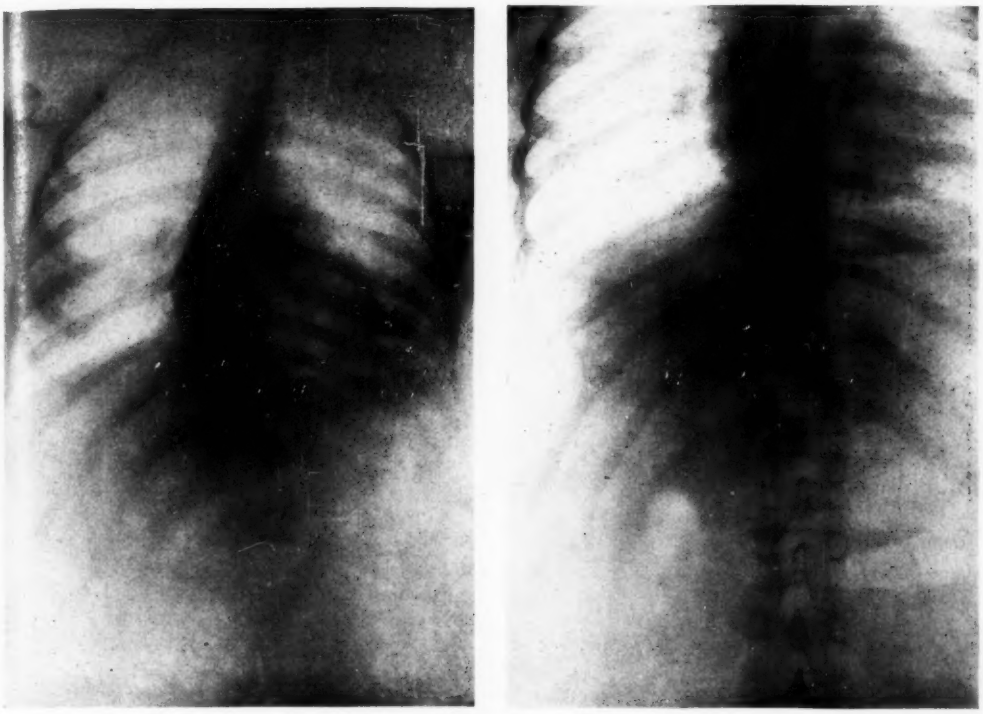


Fig. 9. Mild scoliosis. Shows effect of corrective plaster jacket. a, left. Before treatment. b, In corrective jacket. Note that there is practically a complete correction of the curvature.

formity of the spine. In my own practice I have seen a fairly large number of instances in which not only 2 but even 3 and 4 members of a family have had scoliosis. In a recent study I found an incidence of 10 per cent of hereditary cases. I believe this figure is rather less than the actual because the histories do not always record this detail. Parents so often wish to hide the fact that either of them or any relative has a curvature of the spine.

*Idiopathic scoliosis* (Table II). When all of the known causes of the acquired form of scoliosis have been grouped together they form about 30 per cent of the total number of cases. In the remainder, or 70 per cent, there is no known cause. Consequently they have been grouped under the term of idiopathic, and I believe they must stay in that group until such time as we can identify a definite cause for all or some of them. Some years ago certain schools felt that the vast majority of the acquired scolioses were paralytic in origin. I felt then, and I am certain now, that that is not so, for in the majority of in-

TABLE II.—ETIOLOGY IN 198 CASES OF STRUCTURAL SCOLIOSIS

| | Cases | Per cent |
|---|---|---|
| Idiopathic | 146 | 70 |
| Hereditary | 21 | 10 |
| Paralytic | 12 | 6 |
| Congenital | 9 | 4½ |
| Rachitic | 2 | |
| Vertebral epiphysitis | 1 | |
| Spondylitis | 1 | |
| Pleurisy | 1 | |
| Dislocation of hip | 1 | |
| Arthritis of shoulder | 1 | |
| Defective vision | 1 | |
| Syringomyelia | 1 | |
| Heart disease | 1 | |

This list does not include other well known but infrequent etiological factors.

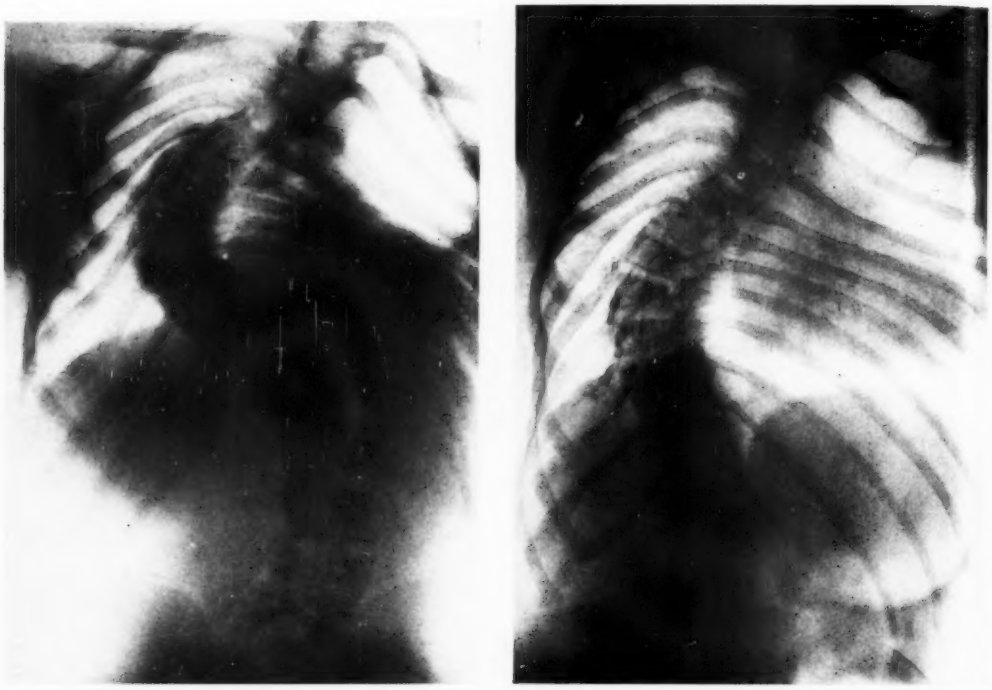


Fig. 10. Severe scoliosis. So called razor back deformity. Spine looks like a reversed question mark. a, left. Note short sharp curve, with extreme reduction in capacity of right side of chest. b, Satisfactory improvement. Patient was treated on convex frame with traction of 103 pounds.

stances there is no history of an attack of infantile paralysis nor is there any evidence of paralysis of muscles anywhere in the body.

### PROPHYLAXIS

Since about 70 per cent of all cases of scoliosis are idiopathic, that is, of unknown origin, there is actually very little one can do to prevent the onset of this deformity. We can, however, frequently prevent an existing scoliosis from increasing. Therefore, if we can manage to get the cases early, much can be done to retain the deformity in its mild state.

In an analysis of 221 private cases I found that the age when the deformity was first noticed was mentioned in 179 cases (Table III). Of this number 118, or 65 per cent, were between the ages of 10 and 14 years. In many of these, perhaps the majority, the deformity was already well established, so that it is reasonable to conclude that the scoliosis had already been in existence for several years. In an additional 27 cases, or 15 per cent, the children were under 10 years of age. We may, therefore, safely assume that in a large majority, at least 80 per cent, the deformity has its origin between the seventh and tenth years. A little reflection will show that this is the period when children, having acquired a measure of independence, being able to bathe and dress unassisted, are no longer under the careful scrutiny of the mother. Much time, even several years, may be lost before the existence of the scoliosis is recognized. It is, therefore, at this age, as we now appreciate it, that the child must be particularly carefully observed.

The means whereby we may discover a scoliosis in its mild stage are the following:

1. Frequent physical examinations. This is possible for the pediatrist, who usually has occasion to see every child under his care one or more times a year.

2. Stout children in whom the adipose tissue may obscure a mild curvature must be examined especially carefully, for such a child may not show any apparent abnormality in the upright position. But if the child is asked to bend forward there will be an easily recognizable asymmetry of the back due to the backward projection of the ribs on the convex side.

3. We should anticipate the appearance of scoliosis in a child who has had an attack of infantile paralysis.

4. We must be on the lookout for and correct physical defects that lead to asymmetry of the trunk such as visual or aural deficiency, torticollis, empyema, or an organic neurologic lesion.

5. All school furniture should be adjustable so that a child may read and write at his desk in a normal position and without undue muscle strain.

6. Particularly careful examination should be made of children in families in which there are some members with scoliosis.

7. Prolonged or exhausting illnesses are likely to be followed by muscle weakness and even muscle imbalance which may lead to scoliotic deformity. A child, recovering from a prolonged illness, ought not to be allowed to undertake severe mental or physical work until he has regained his normal resistance.

### TREATMENT

The treatment of structural scoliosis depends upon the age of the patient, the type and the degree of the curvature. As will be seen from the figures in Figure 1 the large majority of cases are seen in children. Scoliosis in the infant and in the adult constitutes a very special problem and will be taken up separately. In a child the chief factors influencing the mode of therapy are the etiology and the severity of the curvature. As most cases are idiopathic this group will receive attention first.

*Mild idiopathic scoliosis.* The primary concern here is to prevent an increase of the deformity, for, as previously indicated, if the curve remains mild the patient will have no inconvenience, physical or functional. The most effective measure at our disposal to obtain this result is gymnastic exercise. Teschner's system of exercises is an excellent one and is used very widely in our country. The family and the patient must be impressed with the fact that this method of treatment by gymnastic exercises does not operate rapidly and must be continued over a very long period. At times it is helpful to combine with the exercises a light corset of leather or celluloid.

TABLE III.—ANALYSIS OF 221 CASES OF STRUCTURAL SCOLIOSIS

Private patients examined between 1928 and 1937

| | Cases | Per cent |
|---|---|---|
| Total number | 221 | |
| Males | 41 | 18½ |
| Females | 180 | 81½ |
| **Age when deformity was recognized** | | |
| Number of cases in which this information was recorded | 179 | |
| Years | | |
| 0 to 1 | 3 | |
| 1 to 2 | 1 | |
| 2 to 3 | 5 | |
| 6 to 9 | 18 | 10 |
| 10 to 14 | 115 | 64 |
| 14 to 16 | 21 | 12 |
| 18 to 25 | 14 | |
| 34 to 35 | 2 | |
| **Measurement of length of lower limbs** | | |
| Total number | 110 | |
| Limbs of equal length | 102 | |
| Right limb longer than left | 7 | |
| Left limb longer than right | 1 | |

*Moderate idiopathic scoliosis.* The aim of treatment in this group is twofold: reduction of the deformity and maintenance of the correction. In a general way this is accomplished by the use of corrective and retentive apparatus and gymnastic exercise. Many corrective methods are available. Each surgeon should use that procedure with the details of the technique of which he is intimately acquainted. The treatment of scoliosis involves very careful attention to detail, it is time consuming, laborious and requires unusual patience on the part of the surgeon who must continuously exhibit a sympathetic and encouraging spirit. Few men are temperament-

ally qualified to conduct this treatment. Accordingly only those orthopedic surgeons who are specially interested in, and are willing to devote the necessary care to, the treatment of scoliosis ought to engage in it. A carefully planned and properly applied plaster jacket is an effective modality, while an indifferently applied jacket may actually cause an increase of the scoliosis. Making a good plaster jacket is as much a work of experience and expertness as the performance of a major operation.

*Reduction of the curvature.* This can be obtained either through a corrective plaster-of-Paris jacket or by employing traction on a convex frame. The former method is ambulatory and permits the patient to be about and attend school. The latter requires recumbency. The former takes many months of treatment, the latter only a few weeks.

*Application of a corrective plaster-of-Paris jacket* (Fig. 7). The patient is suspended by a Sayre halter until his feet lightly rest on the floor. The traction thus employed serves partially to straighten the spine. The shoulder and pelvic girdles are fixed by lateral bands of canvas or flannel. A lateral traction strap is then placed over the convexity of the curve and attached to a bar on the opposite side of the body. As this band is tightened the spine is further straightened. A large removable pad is placed over the concavity of the back from the spine to the anterior axillary line to provide an opportunity for additional correction of the spine. A plaster-of-Paris jacket is applied. A large window is cut out over the concave side of the curvature. Two small windows are cut out over the convex side; through these thick felt pads are inserted every few days. The pads push the spine over to the concave side and increase the correction. A jacket is left on until the maximum improvement in it is obtained. This usually takes an average of 8 weeks. A new plaster jacket is then applied. Additional jackets are applied until the greatest potential improvement or reduction of the curvature is obtained. This usually takes about 6 months.

*Traction on a convex frame* (Fig. 8). This method is, in my experience the simplest, the fastest, and the most effective means of improving a scoliosis. The patient is placed on a convex frame. A Sayre halter is adjusted at the head of the bed for traction on the patient's head. A pelvic girdle is applied. On each side of this there is attached a band of webbing which extends to the foot of the bed. To each band is attached a rope going over a pulley and holding a weight for traction on the pelvis. Thus we are able to exert longitudinal traction on the spine. As the patient is recumbent, the force of gravity and the superincumbent weight of the body on the spine are removed as deforming elements. Moreover, in the continuous recumbency the muscles relax and the deforming influence of their pull is eliminated. One begins with about 5 pounds of traction on the head and 5 on each side of the pelvis. Each day 1 to 3 pounds are added to each weight, that is, the total pull is increased by from 3 to 9 pounds daily. Lateral traction may be added over the chest at the apex of the convexity. A certain amount of correction is obtained by the attitude of hyperextension and constant pressure on the ribs on the convex side of the curve. In addition expansion of the chest and thorough aeration of the lungs are favored by the use, three or four times a day, of blow bottles. In this method the roentgenogram reveals a remarkably rapid change in the spine. Within 4 to 8 weeks the maximum potential improvement in the curvature is obtained. This treatment has only one possible disadvantage, viz., it is most conveniently carried out in a hospital and therefore it involves the cost of hospitalization and interruption of schooling (Figs. 9 and 10).

When the maximum improvement has been obtained by either the corrective plaster jacket or traction on a convex frame the patient is provided with a corrective or asymmetrical celluloid corset, and a long period of gymnastic exercises is instituted. This part of the treatment lasts at least 2 years. Gradually the corset is left off; at first for several hours each day, later for a day at a time, and finally altogether. The above treatment by either of the corrective measures if carried out uninterruptedly yields satisfactory results in about 80 per cent of the cases. In the remainder in which the deformity increases despite all treatment, a

spine fusion operation is advisable in the hope of checking any further increase of the deformity.

*Severe idiopathic scoliosis.* The cases of severe scoliosis can be divided into those with a stationary deformity and no symptoms and those with increasing deformity with or without symptoms. If the deformity has not increased within several years and the individual has been able to attend to his or her general duties or occupation no treatment is indicated. If, however, the deformity is increasing the treatment should consist of reduction of the curvature as far as may be possible, either through corrective plaster-of-Paris jackets or preferably by traction on a convex frame, followed by a fusion operation on all of the vertebræ in the predominant curve, in the hope that solidification of the spine will stabilize it and prevent further increase of the scoliosis.

*Paralytic scoliosis.* The most favorable time for treatment is during the early stage when the deformity is very mild. If one can decide just what is causing the scoliosis, as for instance a unilateral abdominal paralysis or a shortened limb, this factor should be corrected by a fascial reinforcement of the paralyzed side or a compensatory lift under the short leg. Support of the back should be applied early and maintained for several or even many years in the hope of preventing increase of the curvature. The tendency in paralytic scoliosis is for the curvature continuously to increase often to a very severe degree with so called razor back angulation of the ribs, telescoping of the chest into the abdomen and marked lateral and anteroposterior distortion of the trunk. With this in mind it is probably advisable to fuse the spine in every case of paralytic scoliosis.

*Scoliosis in infants.* The deformity is best controlled by placing the infant on a convex frame with mild traction, or, as Harrenstein[1] suggested, by keeping the infant continuously in a corrective plaster-of-Paris bed. The treatment must be carried out for several years until the spine is either straight or only mildly curved. And even then it is probably advisable for the patient to wear a corset until puberty.

*Structural scoliosis, operative treatment.* There are two types of operation that are frequently employed in the management of scoliosis, viz., a spine fusion and a rib resection.

*Spine fusion.* The purpose of a spine fusion is to obtain a consolidation of the vertebræ in the main portion of the curvature, hoping that thereby the curvature of the spine will not increase. All operations are performed on the posterior arches of the vertebræ. Usually only the vertebræ in the main or primary curve are operated upon. Sometimes, as in poliomyelitis, a more extensive operation is advisable. The operative technical details vary in the hands of different surgeons. The most widely used operation is the Hibbs fusion which includes subperiosteal exposure of the posterior arches, bridging of the interlaminar spaces by segments of bone elevated from the laminæ, contacting of the spinous processes, and a removal of the articular cartilage from the intervertebral articulations. I use the Hibbs technique but have modified it by utilizing a beef bone graft which supports the spine in the corrected position while the fusion of the vertebræ is going on and acts as a frame work for the deposition of new bone.

*Spine fusion* is indicated:

1. In all types of scoliosis in which conservative treatment has failed to control the curvature.
2. In cases in which there is an unequivocal history of progressive increase of the scoliosis.
3. In paralytic scoliosis.
4. In patients with scoliosis who are suffering from persistent and disabling backache.

A spine fusion operation is not a panacea for scoliosis. It is effective in the majority of cases in which it is used. Yet in about 10 or 15 per cent of these cases, even after a spine fusion, the deformity increases. Moreover, the operation is a serious one and therefore should be restricted solely to those patients in whom there is a specific indication and need for it. It is well here to recall that in the vast majority of cases of structural scoliosis, probably in 80 per cent of all cases, conservative treatment is adequate.

[1] Harrenstein, R. J. Die Skoliose bei Saenglingen und ihre Behandlung. Ztschr. f. orthop. Chir., vol. 52.

*Rib resection.* This is an excellent operation for reducing the asymmetry of the back. It is used only in those cases in which there is marked angulation of the ribs. It is a cosmetic operation of considerable merit and particularly applicable to girls or sensitive boys. In this procedure one removes about 5 inches from each of 5 or 6 ribs at the crest of the deformity. Often it can be advantageously combined with a spine fusion, the removed segments of the ribs being used as grafts to unite the vertebræ.

## SUMMARY

Structural scoliosis or rotary lateral curvature of the spine is a comparatively common deformity. The majority of cases are idiopathic, that is, of unknown origin, and develop in the pre-adolescent period; the deformity progressively increases until the end of adolescence and sometimes even in adult life. The deformity affects not only the vertebral column but all of the osseous and soft tissue structures of the trunk including the viscera. In the advanced forms of the deformity, there may be marked functional disturbances. Some cases remain mild; many advance so that there is a conspicuous distortion of the body; not a few result in extreme deformity of the back. Our chief concern is to discover the scoliosis early since at this stage of its development persistent treatment yields the most effective and satisfactory results.

# RENAL FUNCTION TESTS IN THE DIFFERENTIATION OF BRIGHT'S DISEASE FROM SO-CALLED SPECIFIC TOXEMIA OF PREGNANCY

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THE separation of so called specific toxemia of pregnancy from Bright's disease, *antepartum*, is frequently difficult and sometimes impossible, especially when the patient's history is unknown. Peckham and Stout found that 11 per cent of the Johns Hopkins Hospital toxemia cases were wrongly diagnosed while patients were in the hospital.

Van Slyke and his collaborators (20) in an admirable monograph on Bright's disease, conclude that the signs and laboratory findings of aid in diagnosing renal disease are: (a) hypertension, (b) proteinuria, (c) edema, (d) hematuria, and (e) renal functional impairment as shown by tests (urea clearance).

Since pre-eclamptic toxemia fulfills the first four of these five criteria, it seems that the renal function tests offer the best possibility for making the differential diagnosis in cases in which the clinical picture is equivocal.

Will the kidney function tests give normal results in toxemia of pregnancy, thus separating these cases from primary renal disease of measurable degree? This question has been given conflicting answers by different investigators, and can not be regarded as closed.

In the present paper, a large series of cases is analyzed, 5 renal function tests compared, and sources of error considered. Some of the factors responsible for the discordant reports in the literature are discussed.

## MATERIAL

With the exception of a very few private cases, every suspected toxemia patient who entered the hospital between January 1, 1935, and July 1, 1936, has been subjected to 5 routine kidney function tests detailed below. Toxemia was suspected if the blood pressure exceeded 140/90, with or without proteinuria; persistent proteinuria also threw the patient into this class, as did rapid and excessive weight gain associated with toxemic symptoms. When the hypertension or proteinuria were observed only during labor, they were, of course, disregarded. By these criteria, the incidence of toxemia was 7.1 per cent.

The eclamptics were observed during 4 years (from January 1, 1934, to January 1, 1938).

The classification of toxemias adopted is somewhat similar to that of Kellogg, Smith, Teel, and Reid. However, patients who showed only hypertension, transient or persistent, were classed as "hypertension" rather than as pre-eclampsia grade 1. Also some patients were not classified.

## METHODS

*Proteinuria.* Protein was determined qualitatively by the sulphosalicylic acid method. If the reaction was 1+ or more, a quantitative measurement was made by the method of Shevky and Stafford (17).

*Non-protein-nitrogen.* The blood non-protein nitrogen was estimated by the Wong (17) persulphate digestion and direct nesslerization procedure, the Folin-Wu blood filtrate being used.

*Urea nitrogen.* Van Slyke's gasometric urease method was used for all determinations of blood and urine urea (19).

*Phenolsulphonphthalein excretion.* One milliliter of the dye was injected intravenously. Chapman and Halsted's procedure was then followed. The urine was collected at 15 minute intervals for an hour, and at 2 hours. Water was given freely.

*Concentration test.* Fishberg's simple specific gravity test was used. The patient was allowed no fluid after the 5.00 p.m. supper.

From the Department of Biochemistry, Margaret Hague Maternity Hospital.

TABLE I.—THE NORMAL UREA CLEARANCE IN NORMAL PREGNANCY

From the published data of several investigators, and from this series

| Author | Antepartum | | | Puerperium | | |
|---|---|---|---|---|---|---|
| | Cases | Mean per cent | Range per cent | Cases | Mean per cent | Range per cent |
| Nice | 13 | 153 | 80–286 | 13 | 96 | 61–120 |
| Cantarow and Ricchiuti | 39 | 72 | 28–184 | 3 | 121 | 62–184 |
| Dieckmann | 27 | 102 | 45–175 | 10 | 124 | 55–175 |
| Stander, Ashton, and Cadden | 4 | 100 | 92–110 | | | |
| Hurwitz and Ohler | 5 | 127 | 83–161 | | | |
| Cadden and McLane | 9 | 123 | | 11 | 104 | |
| Freyberg, Gillard, and Ganesbauer | 30 | 106 | 77–140 | | | |
| Elden and Cooney | 18 | 85 | 60–118 | | | |
| Present series | 43 | 107 | 60–140 | 84 | 112 | 70–175 |
| Totals and weighted means | 188 | 101 | 28–286 | 121 | 111 | 55–175 |

Urine voided during the night was discarded. Specimens were collected at 8:00, 9:00, and 10:00 a.m. The specific gravities were measured by calibrated hydrometers or by weighing. Correction was made for the protein content. No attention was paid to the diet, a vitiating factor which will be discussed below.

*Urea clearance.* The urea clearance was determined as outlined by Peters and Van Slyke (17). Since many antepartum patients were found to void more urine than could be obtained by catheter, the clearances on these patients were usually done with voided specimens. Postpartum patients still in the hospital were catheterized. Clearances determined in patients back for postpartum study were done with voided specimens. Unless the urine output was in excess of 20 milliliters per hour, third and fourth urines were obtained.

*Urea concentration ratio.* The blood urea was divided into the concentration of urine urea to obtain this ratio. This was done only when the urine output was less than 60 milliliters per hour.

## RESULTS

At the outset, it may be said that the urea nitrogen/non-protein nitrogen is almost worthless in diagnosing renal disease of the degree with which we are largely concerned. The 2 hour and fractional phenolsulphonphthalein excretion do not seem to be of much greater value. The specific gravity test is the most sensitive measure of renal impairment, though unreliable if low results be obtained. The urea clearance is the most generally applicable; the clearance is paralleled by the urea concentration ratio. With all of these tests, except the urea clearance and concentration ratio, subnormal results may—and usually do—mean nothing so far as renal damage is concerned. A low clearance should be checked by one or more repetitions.

*Urea nitrogen/non-protein nitrogen.* Mosenthal and Hiller consider 0.45 as the upper normal for this ratio. Higher values indicate incipient metabolite retention. Many writers have described the decrease in this ratio in normal pregnancy, which means a lower normal than 0.45. The data for the present series have been broken up into several classes, but whatever figure we take as normal, it is evident that abnormal values contribute nothing to the classification of the toxemia. About half of the patients in any group show a ratio above 0.35, while about one-third have ratios above 0.40. The ratio tends to be higher in the Bright's disease group, but few values are outside of the range covered by the other toxemias. *Postpartum* the ratio increases in all groups.

*Two hour phenolsulphonphthalein excretion.* Three-fifths of the nephritics so diagnosed showed subnormal excretion of phenolsulphonphthalein, antepartum (60 per cent is considered as normal). The interpretation of

the low excretion is complicated by the fact that 30 per cent of the patients in the normal, pre-eclamptic, or hypertensive groups have a comparably low excretion antepartum. Postpartum about 1 in 7 of these latter patients fail to excrete as much as 60 per cent of the dye. Thirty-seven per cent of the nephritics give subnormal results after delivery. The range of amounts excreted is similar in all groups, which means that the test is of no value in differential diagnosis.

*Fifteen minute phenolsulphonphthalein excretion.* Chapman and Halsted pointed out that the amount of dye excreted in the first 15 minutes after intravenous injection is a more sensitive renal function test than the determination of the 2 hour total. The fractional phenolsulphonphthalein has been measured in only 67 antepartum and 121 postpartum patients, but some conclusion seems warranted. To conserve space, only the excretion for the first 15 minutes will be considered. This fractional excretion of phenolsulphonphthalein is open to the same objection as is the 2 hour test. Here the shortcoming is even more pronounced. More than half of all antepartum and one-fourth of all postpartum patients fail to excrete as much as 26 per cent of the dye. This applies also in the normal control group. Postpartum the nephritic group as a whole excreted more dye than even the normals. (This failure of excretion is often apparent rather than real, as will be pointed out.)

*Specific gravity test.* Alving and Van Slyke have compared the sensitivity of concentration tests with that of the urea clearance. They found such rigorous tests as the Lundsgaard or Lashmet-Newburgh to be more delicate indicators of renal impairment than the clearance. As we shall see, this also applies in toxemias of pregnancy, although the concentration test used here does not always give reliable results.

Here, as with the phenolsulphonphthalein tests, so many patients (about half) in all groups show subnormal values that the interpretation of a low result in an individual case becomes practically impossible. As a group, the nephritics show much poorer concentrating power than any other group.

Stander, Ashton, and Cadden report that the Mosenthal test is of no value in the differential diagnosis of toxemias.

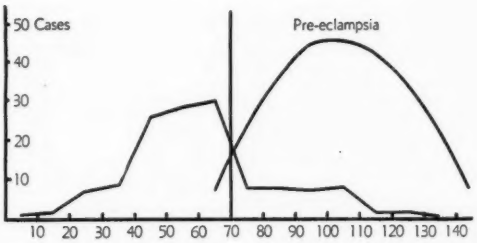


Fig. 1. The distribution of urea clearances in Bright's disease and pre-eclampsia (grade I). Most clearances in the former group are less than 70 per cent, while practically all clearances in the pre-eclamptics are normal.

*Urea clearance.* Van Slyke originally set the normal range of the urea clearance at 80 to 120 per cent. Bruger and Mosenthal suggest 75 per cent as the lower normal limit, 50 to 75 per cent as doubtful, and below 50 per cent definitely abnormal. There is some disagreement as to the effect of normal pregnancy upon the urea clearance. Some of the published data are condensed in Table I. With the methods used in this study, the normal clearance appears to be about the same in late pregnancy and puerperium as outside of pregnancy. However, very high clearances are sometimes seen immediately postpartum and the range seems to be wider than antepartum. Perhaps these higher clearances are attributable to the nitrogen arising from the involuting uterus; high protein intakes are known to increase the clearance, especially after a low protein regimen.

The urea clearances found in pre-eclampsia (grade I) and in Bright's disease complicated by pregnancy are shown in Figure 1. It may be seen at a glance that a differentiation between the two is usually possible by means of this test alone. A mild nephritic may, however, show a normal urea clearance. In such a case the test is useless for diagnosis. The urea clearance findings for all groups are summarized in Table II.

Only in the nephritic group does one find many clearances below 70 per cent (the lower normal suggested by Cadden and McLane and accepted here). The clearances in the pre-eclamptics, eclamptics, and hypertensive pa-

TABLE II.—THE UREA CLEARANCE IN NORMAL AND TOXEMIC PREGNANCY AND PUERPERIUM

The standard deviation was calculated as $\sqrt{\frac{\Sigma \cdot f \cdot (x^2)}{N}}$

| Group | Cases | Mean per cent | Median per cent | Mode per cent | Standard deviation per cent | Range per cent |
|---|---|---|---|---|---|---|
| Normal antepartum | 43 | 107 | 107 | 105 | 22.1 | 60–148 |
| postpartum | 84 | 112 | 113 | 112 | 18.7 | 70–175 |
| Eclampsia antepartum | 19 | 94 | 96 | | 19.0 | 50–128 |
| postpartum | 50 | 96 | 95 | 98 | 18.0 | 34–142 |
| Pre-eclampsia Grade II antepartum | 7 | 98 | 99 | | 23.7 | 44–126 |
| postpartum | 17 | 97 | 94 | | 17.5 | 70–138 |
| Pre-eclampsia Grade I antepartum | 112 | 101 | 102 | 105 | 19.4 | 60–147 |
| postpartum | 147 | 108 | 98 | 95 | 18.6 | 60–180 |
| Hypertension antepartum | 67 | 107 | 106 | 105 | 18.1 | 70–168 |
| postpartum | 86 | 108 | 106 | 104 | 17.7 | 70–190 |
| Unclassified antepartum | 38 | 103 | 104 | | 21.5 | 60–148 |
| postpartum | 41 | 105 | 105 | 103 | 18.9 | 54–152 |
| Bright's disease antepartum | 55 | 59 | 58 | 62 | 20.4 | 20–124 |
| postpartum | 77 | 61 | 50 | 55 | 26.3 | 2–131 |

tients vary around 100 per cent and do not differ significantly from the normal controls. The mean, median, and mode for the nephritics lie at about 60 per cent, which is significantly low.

In all groups, the higher or highest clearance of several shown by a patient was taken as that individual's functional level if certain conditions were fulfilled. If the highest clearance was not checked by a high urea concentration ratio, the average of the two or three highest findings is recorded. If the high clearance were obtained with a blood urea nitrogen which gave a urea nitrogen non-protein nitrogen ratio below that usually shown by the patient, the clearance was disregarded. Clearances of the first and second hours were required to check within 15 per cent. They were not considered if calculated from urine volumes of less than 20 milliliters per hour.

*Urea concentration ratio.* The urea concentration ratio parallels the urea clearance, when the ratio is calculated from low urine volumes. The parallelism is shown in Figure 2 which summarizes the downward course of a patient observed for 10 months. The urea concentration ratio has the same significance as the clearance itself, and often serves as a valuable alternative. The advantage of the clearance is that it can be calculated from any urine volume above 20 milliliters per hour; the urea concentration ratio must be calculated from low volumes.

*Comparison of tests.* The urea clearance and the specific gravity (concentration) tests are compared in Figure 3. The distribution of urea clearances is plotted for all 503 patients showing a specific gravity of 1.020 or higher. Ninety-seven per cent of these clearances are above the lower normal limit of 70 per cent. Of the 3 per cent—14 cases—several clearances are single determinations and are possible low variations. Four are antepartum clearances in patients who showed normal clearances postpartum. The conclusion to be drawn from the figure is that the clearance will very probably be normal until the attainable specific gravity falls below 1.020. With more rigorous concentration tests, the specific gravity limit would be higher. Alving and Van Slyke found the limit to be 1.026 in the Lashmet-Newburgh test. The specific gravity test is, therefore, a more sensitive test for the detection of renal impairment than is the urea clearance. As already mentioned, however, the usefulness of the test is definitely limited in that low results are usually referable to extrarenal factors.

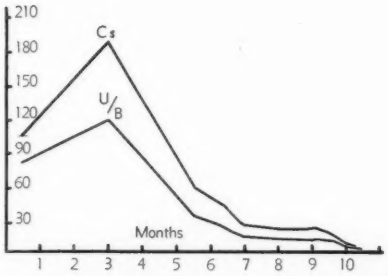


Fig. 2. Parallelism of the urea clearance (Cs) and the urea concentration ratio (U/B). Observations on a case of Bright's disease followed from the beginning of measurable renal impairment to death in uremic acidosis.

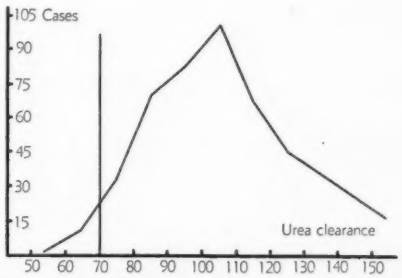


Fig. 3. The distribution of urea clearances in toxemia patients (all groups) who were able to attain a urinary specific gravity of 1.020 or higher. All but 2.8 per cent of the 503 cases had urea clearances of more than 70 per cent.

Figure 4 compares the 15 minute phenolsulphonphthalein excretion with the urea clearance. All patients excreting more than 26 per cent of the dye in the first 15 minutes are represented. About one-fifth of these have subnormal urea clearances. Of the other four-fifths, many would have normal renal function by any test. From this graph we may conclude that the urea clearance is considerably more sensitive than the 15 minute phenolsulphonphthalein.

The other tests, having been shown to be of little value in the toxemias of pregnancy, will not be considered here.

## EVALUATION

*Sequence of tests.* Since the concentration test is the most sensitive of all studied, there would be no indication for doing the others if the toxemia patient should show a specific gravity above 1.022 or even 1.020. This is fortunate, since this is the simplest test of all and may be performed by the practitioner in his own office.

*Sources of error.* About two-fifths of the patients failed to concentrate to 1.022. Alving and Van Slyke have shown that about three-fourths of the specific gravity of urine, above 1.000, is given by salts of which chlorine makes one-third. Urea contributes much to the rest of the significant specific gravity. The quantities of these substances available for excretion are governed by the dietary intake. If the salt and protein be restricted, the urine can not show so high a specific gravity as it otherwise would.

Most of these toxemia patients had been on a low-salt diet for days or even weeks before some of the concentration tests were done. Many were on a low protein intake as well. Still a third factor which will reduce the attainable specific gravity is the dilution of the urine by edema fluid, which is found in many toxemia patients. These factors will also reduce the attainable specific gravity of the urine in normal subjects. The control series of normal pregnant women were kept on this diet for 2 or more days before the concentration test was done. As was seen above, nearly two-thirds of antepartum and two-fifths of postpartum normals failed to concentrate to 1.022.

As for the phenolsulphonphthalein tests, there is a remarkable uniformity in the proportion of patients failing to excrete the normal amount of dye (52 to 60 per cent in each group). This suggests a common factor. One consideration is undoubtedly the dilated

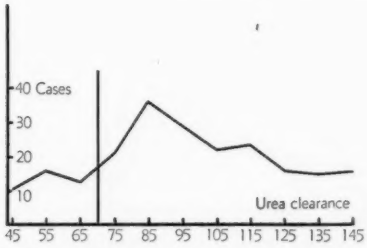


Fig. 4. The distribution of urea clearances in toxemia patients (all groups) who had a 15 minute phenolsulphonphthalein excretion of 26 per cent or more. One-fifth of these patients had urea clearances of less than 70 per cent.

TABLE III.—UREA CLEARANCES FOUND IN THE FOLLOW UP OF PATIENTS DIAGNOSED AS NEPHRITICS ON THE BASIS OF LOW UREA CLEARANCES ANTEPARTUM OR IN PUERPERIUM

**Hypertension, proteinuria, and edema persistent**

| Antepartum per cent | Puerperium per cent | 6 wks. per cent | 3 mo. per cent | 6 mo. per cent | 1 yr. per cent | 2 yrs. per cent | Remarks |
|---|---|---|---|---|---|---|---|
| 41 | 29 | 26 | 16 | Dead | | | Uremia 4 mo. postpartum |
| 39 | 40 | | Dead | | | | Uremia 3 mo. postpartum |
| 37 | 24 | 13 | Dead | | | | Uremia 3 mo. postpartum |
| 61 | 60 | | | Dead | | | Uremia and cardiac |
| 58 | Dead | | | | | | Benign nephrosclerosis |
| 28 | 40 | 50 | | 52 | | Dead | Uremia |
| 49 | 80 | | 26 | | | | Severe hypertension |
| | 65 | | | 61 | | | Anemia, subjective |
| | 8 | 21 | 37 | 35 | 50 | 46 | History |
| | 67 | 62 | | 57 | 48 | | Subjective |
| 33 | 71 | 43 | 54 | 65 | 67 | 56 | Subjective |
| 64 | | | | | 59 | 31 | Hematuria, subjective |
| | 38 | | | 42 | 46 | 51 | Eclampsia twice |
| | 54 | | 45 | | 46 | | Eclampsia |
| | 68 | | | | 52 | | Gross proteinuria |
| | 63 | 57 | 69 | 69 | 45 | | Subjective |
| 60 | 57 | | | | 64 | 54 | Subjective |
| 64 | 69 | | | 54 | | | Subjective |
| 30 | 25 | | 24 | 28 | | | Subjective |
| 48 | 47 | | | 84 | | 77 | Hematuria |

**Hypertension and proteinuria persistent**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 57 | 60 | | 49 | 55 | | | History, hematuria |
| 45 | 59 | 58 | 49 | 54 | 57 | | Severe hypertension |
| | 57 | | 42 | | | | Hematuria |
| | 62 | | | | | 60 | History |
| 52 | 67 | 66 | | | | | Hematuria |
| 53 | 51 | | | 43 | | | History, subjective |
| 40 | 40 | 48 | 45 | 64 | | | Repeated toxemia |
| 52 | 39 | | | 28 | 31 | 32 | Chr. pyelonephritis |
| 58 | 55 | | | 40 | | | No complaints |
| 53 | 30 | | 39 | 36 | | | No complaints |
| 65 | 66 | | | 57 | | | Severe hypertension |

**Proteinuria persistent**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | 56 | | | 64 | | | No complaints |
| 64 | | | | 67 | 64 | | Edema, subjective |
| | 43 | 50 | 52 | 61 | | | Acute "pyelitis" |
| 34 | 42 | 50 | 46 | | | | Edema, high cholest. |
| 61 | 66 | 67 | 69 | | | | Edema, subjective |

TABLE III.—UREA CLEARANCES FOUND IN THE FOLLOW UP OF PATIENTS DIAGNOSED AS NEPHRITICS ON THE BASIS OF LOW UREA CLEARANCES ANTEPARTUM OR IN PUERPERIUM—Continued

Neither hypertension nor proteinuria persistent

| Antepartum per cent | Puerperium per cent | 6 wks. per cent | 3 mo. per cent | 6 mo. per cent | 1 yr. per cent | 2 yrs. per cent | Remarks |
|---|---|---|---|---|---|---|---|
| | 60 | | | | | 38 | Hematuria, edema |
| 44 | | 45 | 59 | 58 | 45 | 60 | History, hematuria |
| 44 | 48 | 27 | | 43 | 54 | | Chr. pyelonephritis |
| | 45 | 58 | | 69 | | | Repeated toxemia |
| 66 | 63 | 34 | | 49 | | | No complaints |
| | 66 | | | 69 | | | History, subjective |
| | 43 | | | 56 | 62 | | No complaints |
| 68 | 68 | 69 | | | 94 | | Repeated toxemia |
| | 45 | | | 32 | 38 | 32 | Occasional proteinuria, edema |
| 46 | 43 | | | | 37 | 45 | Hematuria, anemia |

ureters which cause a lag in the appearance of an excreted foreign substance in the bladder.

The standard urea clearance can not be calculated from urine volumes of less than about 20 milliliters per hour (Chesley, 6, 7). This has apparently been a source of error in several publications. For instance, Dieckmann routinely did urea clearances on urine specimens obtained during a concentration test. Many of these urine volumes were below the critical limit of 20 milliliters per hour; since the fundamental assumption on which the standard clearance is calculated does not hold for this range of volumes, he did not really calculate urea clearances. This is probably enough to account for his finding that the "urea clearance" averaged about 50 per cent in all toxemic patients, and for his conclusion that the "urea clearance is of little value in pregnancy in the majority of cases as an aid in diagnosis or prognosis." Even without this consideration, Dieckmann's results do not appear convincing. For instance, from his Table V it seems that the urea clearance is less than 39 per cent in two-thirds of toxemic multiparæ having no history of toxemia. One-fourth of this group had clearances of less than 19 per cent. Yet the blood urea values were normal, the mean for this group being $15.4 \pm 0.74$.

Several investigators have reported low urea clearances in eclampsia. None of these gives actual urine volumes or makes any mention of the vitiating factor of oliguria. Hurwitz and Ohler do say that a clearance of 8 per cent was observed in an eclamptic who was almost anuric. They also comment upon the rapid rise in urea clearance as the toxemia regresses (and therefore as the oliguria clears). In the present series, urea clearances were done antepartum or immediately postpartum in 68 eclamptics. Only 4 of these clearances were less than 70 per cent. Three of the 4 patients have been shown in the follow-up to have a measurable and probably permanent renal impairment. In all of the 68 cases, the clearances were calculated from urine volumes of more than 20 milliliters. The use of smaller volumes on the same day gave erroneous calculations in accord with some of the published data mentioned above.

*The urea clearance in differential diagnosis.* The data presented here seem to show that the urea clearance will often differentiate Bright's disease complicated by pregnancy from so called specific toxemia. Bright's disease as used here is a very general term which includes glomerular (hemorrhagic) nephritis, pyelonephritis, advanced benign and malignant nephrosclerosis, and even one known case of polycystic kidney. In short, any renal impairment of measurable degree has been called Bright's disease. Following a bad obstetrical precedent, "nephritis" has been occasionally used as a synonym. Cases conforming to the usual criteria for Bright's

disease, before the pregnancy, have been included though in some no renal impairment was shown by the tests used. An effort has been made to differentiate cardiovascular hypertension without functionally detectable nephrosclerosis. In many cases, the follow-up has resulted in changed diagnoses.

In how many cases diagnosed as nephritis by the urea clearance can the diagnosis be sustained? The value of the test stands or falls on the answer to this question.

During the 18 months from January 1, 1935, to July 1, 1936, urea clearances of less than 70 per cent were observed in 119 patients. In 42 of these cases, no effort was made to follow up the patient's renal function because the test was not regarded as significant for one or more of several reasons. In many instances, immediate repetition of the clearance gave normal values, indicating that the one reading below 70 per cent was a low variation in a normal kidney. If a single clearance was done, and this was less than 70 per cent, the clearance was disregarded if the specific gravity of the urine exceeded 1.022 or if the urea concentration ratio was above 70.

Five other cases with urea clearances of less than 70 per cent have been thrown out because these patients had acute pyelonephritis from which they have apparently recovered.

This leaves 72 cases with low clearances. Of these, 20 could not be followed up for various reasons. The 52 remaining cases have been followed for periods up to 3 years.

In Table III are shown the results of the follow-up study in 48 cases. In all but 2 of these, urea clearances have continued at a low level for as long as the patient was observed (to date). Both hypertension and proteinuria have persisted in more than half. Of the 4 cases not shown, it is considered that the diagnosis of nephritis is justified by the history in 2 cases. One other case had a nephropexy at 7 months postpartum; the granular kidney observed at that time, plus a mild hypertension and proteinuria are enough to confirm the diagnosis here. In the other, urea clearances were not obtainable in the follow-up, but moderate hypertension (190/110), gross proteinuria, edema, hematuria, and final death in uremic acidosis were taken as proof of renal disease.

Six of the patients are dead. Five died in uremic acidosis, and the sixth of heart failure.

## SUMMARY AND CONCLUSIONS

1. Renal function has been studied in 599 cases of toxemia of pregnancy. A control group of 119 patients was also studied. The tests used were the urea nitrogen/non-protein nitrogen, the 2 hour and the fractional excretion of intravenous phenolsulphonphthalein, the Fishberg concentration test, the urea clearance, and the urea concentration ratio.

2. The toxemias were classified as follows: eclampsia, 79 cases; pre-eclampsia grade I (mild), 217 cases; pre-eclampsia grade II (severe), 20 cases; hypertension, 125; unclassified, 67; and Bright's disease, 91.

3. The urea nitrogen/non-protein nitrogen, the 2 hour, and fractional phenolsulphonphthalein excretion tests are apparently not very sensitive, and are influenced by several extrarenal factors. They are not of much value in differentiating nephritis from so called specific toxemia of pregnancy.

4. The specific gravity test is the most sensitive studied. If the specific gravity should be as high as 1.022, there is apparently no indication for doing the other tests, which would all be normal—except for extrarenal factors.

5. Many patients, even without toxemia, will fail to concentrate to 1.022 when salt and protein of the diet are restricted.

6. The urea clearance is the most generally applicable of all tests studied. The level in normal pregnancy is about the same as out of pregnancy.

7. In eclampsia, pre-eclampsia, and hypertension the urea clearance does not differ significantly from values observed in normal patients.

8. In Bright's disease complicated by pregnancy, the clearance is often less than 70 per cent. This differentiates renal disease of measurable degree from specific toxemia of pregnancy.

9. The urea concentration ratio parallels the urea clearance, and at very low urine volumes is a valuable alternative.

10. Some contradictory results in the literature, especially regarding urea clearance findings, are discussed.

11. A follow-up of patients having urea clearances of less than 70 per cent shows that the function does not improve. In most cases hypertension or proteinuria or both persist. Edema and hematuria are often found. Therefore, diagnoses of Bright's disease made on toxemic patients showing lowered clearances are justified.

I am greatly indebted to Drs. S. A. Cosgrove, J. F. Norton, and E. G. Waters for reading and criticizing the manuscript. Most of the patients were on the services of these men. The majority of the blood non-protein nitrogen determinations were done by Miss P. Brett, while most of the phenolsulphonphthalein and concentration tests were done by the Misses M. Wolfe and F. Orsato. The attending, house, and nursing staff have co-operated throughout.

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# THE BLOOD VOLUME AND HEMOGLOBIN AFTER TRANSFUSION

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IN an early paper, Boycott and Douglas showed that the blood volume of rabbits following transfusion apparently returned to the pre-transfusion level the next day after the transfusion. However, in a later work, Boycott and Oakley found that when they corrected the figure for blood volume there occurred an increase in the blood volume of the rabbits that were transfused, which was practically equivalent to the cell volume of the blood given as a transfusion. This indicated that the plasma volume tended to remain at a constant level and the changes that occurred in the blood volume depended on the changes in the cell volume of the host. That the plasma volume remains constant under varying conditions has been demonstrated by Rowntree, Brown, and Roth and by Bock.

Working on rabbits, Boycott and Oakley found, also, that the value for total hemoglobin was at a lower level than it had been calculated to be 24 to 72 hours after transfusion. Essentially the same condition was found to exist by Krumbhaar and Chanutin who produced experimental plethora in animals by repeated transfusions. These two groups of investigators found that the value for hemoglobin after transfusion was never so great as it was calculated to have been and that some of the hemoglobin was lost from the circulation after transfusion.

It has been demonstrated that reactions to blood transfusion produce a deleterious effect on the otherwise beneficial effects of blood transfusion on the content of hemoglobin in the blood of the recipient (9). At the time of the study of the effects of reactions on the concentration of hemoglobin of the blood following transfusions, certain data were obtained which allowed calculation of blood volume of the recipients of those transfusions. The effects of transfusion on the blood volumes of the recipients thus were determined. At the same time, the changes in the values for total hemoglobin were observed.

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## METHOD

All transfusions, in this work, were given by the citrate method of Lewisohn, as described by Lundy, which is the routine method of blood transfusion at the Mayo Clinic. Each transfusion consisted of 500 cubic centimeters of blood and 110 cubic centimeters of physiologic solution of sodium chloride and sodium citrate, making a total of 610 cubic centimeters of fluid. The 610 cubic centimeters of blood, saline, and citrate were administered in a period of 30 minutes at each transfusion.

All hemoglobin determinations were made on the Cenco-Sheard-Sanford "photelometer." The technique for determining the value of hemoglobin was applied as described by the inventors of the photelometer (8). The concentration of hemoglobin of the donors was determined at the time that the blood was being withdrawn for transfusion. The concentration of hemoglobin of the recipients of the transfusions was determined immediately before transfusion, 3 minutes after transfusion, and, again, at the end of the first, second, fourth, sixth, eighth, and tenth days after transfusion.

Data concerning body weight were available in 84 cases. Knowledge of body weight made it possible to estimate the blood volume of these 84 patients, using the figure 82.8 cubic centimeters of blood per kilogram of body weight (the blood volume of 82.8 cubic centimeters is the average volume per kilogram of body weight given by Rowntree, Brown and Roth).

The total volume and the total content of hemoglobin of each transfusion being known,

TABLE I.—VALUES FOR HEMOGLOBIN AND CHANGES IN BLOOD VOLUME AFTER BLOOD TRANSFUSION; NINETY-NINE CONSECUTIVE CASES; GENERAL RUN OF CASES; AVERAGE MEAN VALUES

| | Before transfusion | After transfusion | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | 3 minutes | 1 day | 2 days | 4 days | 6 days | 8 days | 10 days |
| Value for hemoglobin, gm. per cent | 9.40 | 10.00 | 10.70 | 10.90 | 10.50 | 10.90 | 10.30 | 10.30 |
| Increase in value for hemoglobin, gm. per cent | | 0.60 | 1.30 | 1.50 | 1.10 | 1.50 | 0.90 | 0.90 |
| Increase in total hemoglobin, gm. (calculated) | | 77.8 | 77.8 | 86.6 | 68.9 | 86.6 | 50.2 | 50.2 |
| Total hemoglobin, gm. (calculated) | 392.9 | 470.7 | 470.7 | 479.5 | 461.5 | 479.5 | 443.1 | 443.1 |
| Blood volume, c.cm. | 4,167 | 4,695 | 4,399 (gain 232 c.cm.) | | | | | |

it was possible to calculate the approximate amount of change in the volume of blood of the recipient of such a transfusion. In this group of 84 cases, it was found that, in the period of 30 minutes required to administer each transfusion, approximately 82 cubic centimeters of fluid disappeared from the circulation of the recipients of this mixture of 610 cubic centimeters which included blood, saline and citrate. Consequently, for the benefit of accuracy, it was necessary to subtract this amount of 82 cubic centimeters from the total volume of 610 cubic centimeters added with each transfusion, because the second determination of concentration of hemoglobin was not made until all the blood was run into the vein of the recipient. This means that the blood volume of the recipient at the end of the transfusion of the 610 cubic centimeters actually was increased by only 528 cubic centimeters.

The concentration of hemoglobin at the various intervals of observation having been determined and the volume of fluid added by each transfusion having been established as 528 cubic centimeters, it was possible to calculate, in the following manner, the total volume of blood of patients being transfused:

Let $x$ = the blood volume before transfusion
Let $y$ = the value for total hemoglobin before transfusion
Volume × concentration = total amount,
then
$(x) \times \text{concentration} = y$
or

$$\frac{y}{x} = \text{concentration}$$

and after blood transfusion

$$\frac{y + \text{gm. hgb. added}}{x + \text{volume of transfusion}} = \text{new concentration.}$$

Example:

concentration hgb. immediately before transfusion = .094 gm./c.cm.
concentration hgb. immediately after transfusion = .100 gm./c.cm.
total hgb. added by transfusion = 77.8 gm.
total (corrected) volume of transfusion = 528 c.cm.

substituting in the formula:

$$\frac{y}{x} = 0.094 \div y = 0.094x$$

and

$$\frac{y + 77.8}{x + 528} = 0.100 \div \frac{.094x + 77.8}{x + 528} = 0.100$$

then

$$0.094x + 77.8 = 0.100x + 52.8$$

or

$$0.006x = 25$$
$$x = 4167$$

Therefore, the blood volume before transfusion was 4167 c.cm.

Again,

$$y = 0.094x \div y = 0.094\ (4167)$$
$$y = 392.9$$

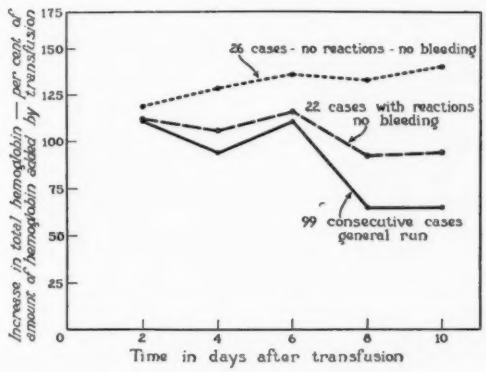


Fig. 1. Graphic illustration of comparative results in three groups of cases studied.

TABLE II.—VALUES FOR HEMOGLOBIN AND CHANGES IN BLOOD VOLUME AFTER BLOOD TRANSFUSION; TWENTY-TWO CASES; REACTIONS TO TRANSFUSION; NO BLEEDING; AVERAGE MEAN VALUES

| | Before transfusion | After transfusion | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | 3 minutes | 1 day | 2 days | 4 days | 6 days | 8 days | 10 days |
| Value for hemoglobin, gm. per cent | 8.44 | 9.41 | 9.41 | 10.04 | 9.94 | 10.14 | 9.53 | 9.57 |
| Increase in value for hemoglobin, gm. per cent | | 0.97 | 1.36 | 1.60 | 1.50 | 1.70 | 1.09 | 1.13 |
| Increase in total hemoglobin, gm. (calculated) | | 94 | 94 | 104.8 | 100.3 | 109.2 | 82 | 83.8 |
| Total hemoglobin, gm. (calculated) | 344.6 | 438.6 | 438.6 | 449.4 | 444.9 | 453.8 | 426.6 | 428.4 |
| Blood volume, c.cm. | 4,083 | 4,627 | 4,476 (gain 392 c.cm.) | | | | | |

Therefore, the value for total hemoglobin before transfusion was 392.9 gm.

The blood volume immediately after transfusion was, thus,

$$4167+528=4797 \text{ c.cm.}$$

and the value for total hemoglobin immediately after transfusion was

$$392.9+77.8=470.7 \text{ gm.}$$

Thus, having established certain facts about the blood volume and the value for the hemoglobin of the recipient's circulating blood, it was then a matter of determining changes in concentration in order that the changes in blood volume in the next 24 hours could be calculated, for example:

Let $x$ = the new blood volume
$(x) \times$ concentration = total amount
The total amount of hemoglobin = 470.7 gm.
The new concentration = 0.107 gm./c.cm.
then,

$$(x) \times 0.107 = 470.7$$
$$x = 4399$$

Therefore, the blood volume at the end of 24 hours was 4,399 cubic centimeters. This represents a loss of 306 cubic centimeters of the volume of 528 cubic centimeters added by transfusion, or a gain of 232 cubic centimeters in the total blood volume of the recipient by transfusion. This gain represents approximately the cell volume of 500 cubic centimeters of blood of the average person. The results of these observations and calculations are expressed in Tables I, II, and III, and Figure 1.

## OBSERVATIONS

The behavior of the blood volume and hemoglobin in a group of 99 persons following transfusion of 500 cubic centimeters of citrated blood is expressed in Table I. In this table, it is seen that the blood volume was calculated to be 4,167 cubic centimeters before transfusion. Twenty-four hours after the transfusion it was found to be 4,399 cubic centimeters. This change represents a gain of 232 cubic centimeters in total blood volume, which is equivalent to 46.4 per cent of the volume of the blood added. This means that the volume has increased in an amount exactly equivalent to the cell volume contained in 500 cubic centimeters of blood of the average normal person. This same table illustrates, also, that the total hemoglobin apparently increased by an amount equal to that added in the transfusion. This gain was maintained for several days and dropped off to almost half the amount gained by transfusion. This drop is undoubtedly owing to bleeding and to reactions following transfusion as will be evident on examination of Tables II and III.

Table II depicts the behavior of the hemoglobin and the changes in the blood volume following transfusion of 500 cubic centimeters of citrated blood in a group of 22 persons in whom no bleeding was evident, but who did have reactions to transfusion. A reaction was said to have occurred when the patient had chills and elevation of temperature above normal to at least 100 degrees F. (37.8 degrees C.) or higher. This table illustrates that the blood volume had increased 392 cubic centimeters by the end of the first day after transfusion. This was 70 per cent of the volume of the blood added by transfusion. Apriori, we would expect this to increase approximately 48 per cent of the 500 cubic centimeters volume of the blood added by transfusion. The probable reason why the blood volume, in this instance,

TABLE III.—VALUES FOR HEMOGLOBIN AND CHANGES IN BLOOD VOLUME AFTER BLOOD TRANSFUSION; TWENTY-SIX CASES; NO REACTIONS AND NO BLEEDING; AVERAGE MEAN VALUES

| | Before transfusion | After transfusion | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | 3 minutes | 1 day | 2 days | 4 days | 6 days | 8 days | 10 days |
| Value for hemoglobin, gm. per cent | 8.75 | 9.55 | 10.04 | 10.43 | 10.65 | 10.61 | 10.75 | 10.87 |
| Increase in value for hemoglobin, gm. per cent | | 0.80 | 1.29 | 1.68 | 1.90 | 2.06 | 2.00 | 2.12 |
| Increase in total hemoglobin, gm. (calculated) | | 79.7 | 79.7 | 94.2 | 102.3 | 108.2 | 106.1 | 110.6 |
| Total hemoglobin, gm. (calculated) | 293.3 | 373 | 373 | 387.5 | 395.6 | 401.5 | 399.4 | 403.9 |
| Blood volume, c.cm. | 3,363 | 3,891 | 3,715 (gain 352 c.cm.) | | | | | |

remains at such a high level of increase after transfusion is that there were several transfusions of polycythemic blood having a concentration of hemoglobin of as much as 22.7 grams per cent. The cell volume was not determined but it is reasonable to assume that it was high for this group. Also, Table II illustrates that the content of hemoglobin of the recipient increased slightly for several days above the amount added by transfusion, but by the end of the eighth day it had decreased to a point below the amount added by the transfusion and remained at that level. This represents a loss of about 10 per cent of the amount of hemoglobin added by transfusion.

Table III reveals the nature of the changes that occurred in the blood volume and in the behavior of the content of hemoglobin of the blood of the recipients of transfusions who did not give evidence of reaction to transfusion or signs of bleeding after transfusion. This table illustrates that the volume of blood increased 352 cubic centimeters which is 66 per cent of the volume of the blood transfusions. The total hemoglobin of the blood of the recipients increased in a constant progressive manner above the amount added by the transfusions for the period of 10 days during which the amount and concentration of hemoglobin of these patients were followed. At the end of the tenth day after transfusion, the value of hemoglobin of the recipients in this group had increased 40 per cent above the amount added by the transfusion.

Comparing the results in the three tables it is seen that the blood volume increased, in each instance, to a point approximately equivalent to the cell volume of the blood added by transfusion. Also, these tables illustrate that the values of total hemoglobin after transfusion behaved according to the conditions that existed after the transfusion. Thus, it was observed that, if bleeding and reactions occurred after transfusion in the general run of cases, the value for hemoglobin increased to an amount about equal to that added by the transfusion, only to decrease to 65 per cent of the amount added by the transfusion at the end of the eighth day. If reactions occurred after transfusion and bleeding did not occur, the total hemoglobin increased above the amount added by transfusion until the eighth day, at which time it decreased to 89 per cent of the amount added by transfusion. If both reaction and bleeding did not occur, the total hemoglobin increased progressively until the tenth day, at which time it was 40 per cent above the amount added by transfusion. Also, the increase in total hemoglobin of the group of patients who had no reactions was 50 per cent more than the increase shown on the tenth day by the patients who had reactions. (For comparison, see Figure 1.)

## SUMMARY

The method of calculating blood volume has been explained and examples have been included.

It was found that, among human beings who have secondary anemia, the blood volume increased in an amount approximately equivalent to the cell volume of the blood added by transfusion.

Among human beings who have secondary anemia, the total hemoglobin after transfusion apparently increases above the amount added by transfusion during the 10 day period following blood transfusion, provided that there

has been neither reaction to transfusion nor evidence of bleeding after transfusion.

Reactions occurring after transfusion tend to reduce the total hemoglobin added by transfusion, so that, at the end of 10 days, the total hemoglobin is but 90 per cent of the amount added by transfusion. This figure at the end of the tenth day is only 50 per cent as great as that of the patients who did not have reactions.

These observations uphold the conception that the plasma volume tends to maintain a constant level and that changes in blood volume are brought about by changes in the cell volume only.

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# CLINICAL SURGERY

*FROM ST. MARY'S HOSPITAL, LONDON*

## THE TECHNIQUE OF GASTRODUODENECTOMY

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IN the surgery of simple ulcer of the stomach or duodenum one fact stands out starkly clear. If the ulcer together with a considerable area of the pyloric end of the stomach including the sphincter be removed, the physiological conditions prevailing in the stomach are so altered that a recurrence of the ulceration, while it is not unknown, is extremely unlikely to happen. In the surgical unit at St. Mary's Hospital, London, this principle is adhered to whenever possible. The restitution of the alimentary canal is effected by implanting the stomach laterally into the upper part of the jejunum brought up in front of the splenic flexure of the colon which lies very deeply in the abdomen (Fig. 1). Axial union of the stomach to the duodenum has been carried out 42 times in the case of duodenal ulcer with 3 deaths (7 per cent) and 28 times in the case of gastric ulcer with 1 death (3.6 per cent). This method of restoration is attractive from a physiological point of view, but the convalescence is not so smooth, being sometimes disturbed by temporary acute dilatation of the stomach, and occasionally there is a little subsequent digestive discomfort which may, perhaps, be related to the loss of mobility of the organ due to the small remaining segment being sutured to the fixed duodenal stump. Hence lateral implantation into the jejunum has become the standard method. In 116 gastroduodenectomies done in this way for duodenal ulcer only 1 death occurred (0.86 per cent). It was due to an injury to the pancreas. In 66 resections for gastric ulcer the mortality has unfortunately been high, 4 deaths (6 per cent). One of these occurred 4 months after the operation from a lung abscess. One was due to peritonitis from a leak at the suture line, one to peritonitis with intact suture line, and another to hemorrhage from a vessel in the cut end of the stomach. The operation has been carried out on weakly, wasted, often anemic individuals and sometimes after 60 years of age.

### THE OPERATION FOR DUODENAL ULCER

The abdomen is opened by a right paramedian incision, extending from the neighborhood of the ensiform cartilage to the right of the umbilicus. The rectus muscle is retracted laterally to gain access to the peritoneum.

The duodenum is inspected to determine whether or not excision is possible. Very large ulcers surrounded by massive callous inflammatory tissue which extends so far down the duodenum that the surgeon feels he cannot reach normal intestine for suture are best left alone and treated by gastrojejunostomy. However, if, by careful dissection, the duodenum is freed from adhesions to the gall bladder and other neighboring parts, and particularly if the layer of newformed tissue over the descending part of the duodenum, so often present, be removed, it will often be found possible to excise ulcers which at

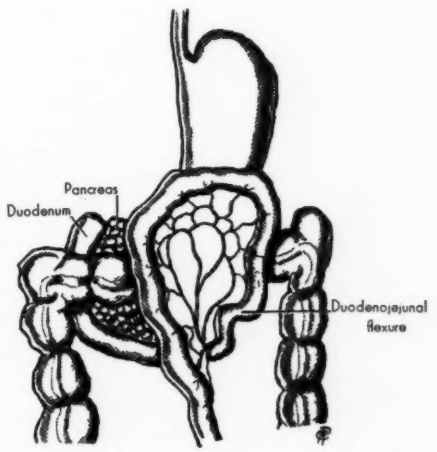


Fig. 1. Method of restoring continuity of alimentary canal.

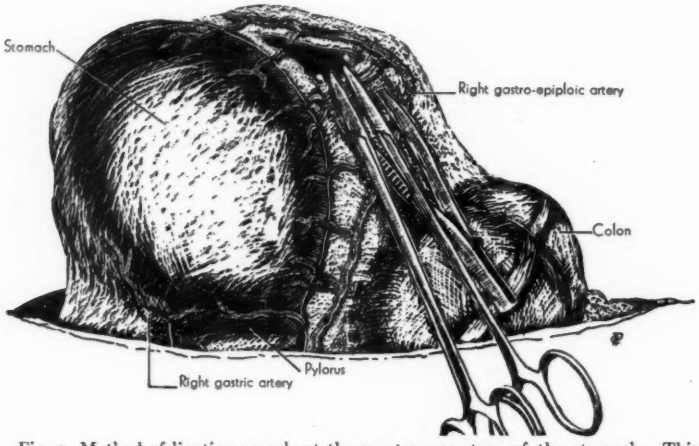


Fig. 2. Method of ligating vessels at the greater curvature of the stomach. This and subsequent diagrams are drawn from the viewpoint of the surgeon standing to the right of the patient.

first sight look quite unresectable. Next, at about the middle of the greater curvature of the stomach below the gastro-epiploic artery, an aperture is torn in the thin omentum so that the lesser sac is penetrated and the posterior aspect of the duodenum can be examined. There is usually an ulcer here. The extent of invasion of the pancreas is estimated. This preliminary review having shown that resection can be carried out, the vessels of the greater curvature are ligated between the stomach and the gastro-epiploic artery. The manner of doing this is shown in Figure 2. Fine No. 00 catgut is used for these ligatures. A length of about 2 inches of the greater curvature is thus bared at about 1½ inches from the pylorus. In a difficult case a similar short length of the lesser curve is ligated off from the gastrohepatic omentum. Two large clamps are then placed across the stomach in this area and the organ cut across between them (Fig. 3). This procedure allows of easier access to the posterior

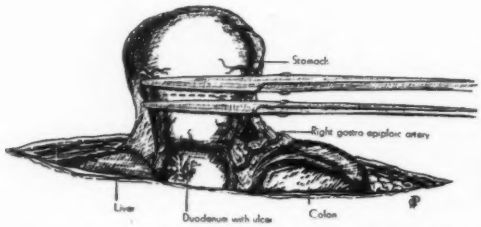


Fig. 3. Line of section of stomach in order to facilitate access to a posterior duodenal ulcer.

aspect of the duodenum and greatly facilitates the removal. Now, little by little, the lower and upper borders of the duodenum are freed to a point distal to the ulcer. Every small vessel must be seen and divided between clamps as it is met. The really difficult part of the operation is on the pancreatic side. Many small vessels come off the pancreaticoduodenal artery which runs in the groove between the pancreas and duodenum. The field must be kept bloodless if possible. Cicatrization may bring the common bile duct in close proximity to the duodenum at its upper border. It is in danger of injury here. In order to avoid damage to the pancreas, which may be fatal, or to the common bile duct, the dissection must be kept close to the duodenal wall. Figure 4 represents the dissection completed. If the ulcer has penetrated into the pancreas the base of the ulcer must be left on this viscus untouched. During the dissection the margin of the duodenal wall will be separated from the pancreas and the cavity of the duodenum opened into. It is sometimes rather tedious to find the right layer of separation beyond the ulcer base, but it can nearly always be done (Fig. 5). A narrow clamp is then placed just beyond the ulcer and the diseased part of the duodenum removed by the cautery (Fig. 6). The duodenal stump is closed by two layers of sutures as shown in Figures 7 and 8. Occasionally a reinforcing stitch may be necessary but the omentum is rarely sewed over it. A duodenal fistula has never occurred in this series. On two occasions it seemed almost impossible to arrive at normal duodenal wall beyond the distal

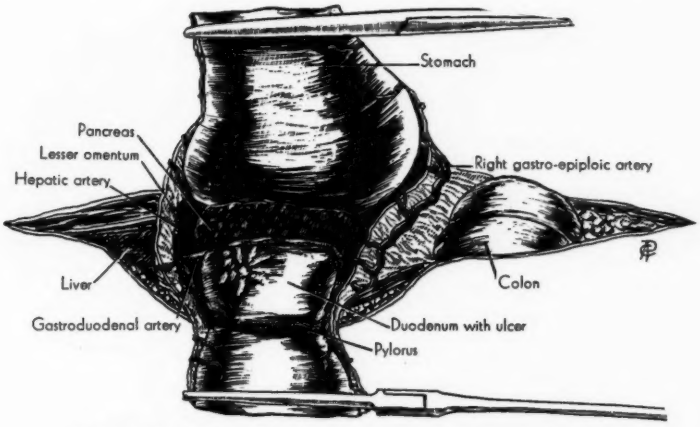


Fig. 4. The duodenum has been dissected free beyond the ulcer.

edge of an ulcer penetrating the pancreas. In both cases the anterior duodenal wall was folded in and sutured down over the ulcer, the stitches going through the fibrous tissue round the margin of the ulcer. The whole ulcer base was buried in this way and peritoneal surfaces brought into contact with each other. It is dangerous to pass sutures through normal pancreatic tissue, but safe to insert them through the fibrosed destroyed pancreatic tissue immediately surrounding the ulcer. Once, by mistake, in a very extensive ulcer, the common pancreatic and bile duct was cut right across. The cut surface of the gland with the open duct was successfully implanted into the open end of the duodenum.

The surgeon now returns to the proximal part of the stomach and ligates the vessels along the greater and lesser curvatures until he deems a sufficient length has been denuded. It is not possible to estimate accurately the proportion of the stomach removed: the area taken away varies. In length it amounts to 4 or 5 inches approximately and depends upon the amount of dilatation of the stomach which may have taken place. When a sufficient area has been devascularized the first part of the jejunum is drawn up from below the colon and fixed by traction sutures transversely across the posterior aspect of the stomach, the proximal end of the loop being attached to the greater curvature. The two viscera are joined together by a thin catgut, running seromuscular suture. About 4 or 5 inches of jejunum should exist between the duodenojejunal flexure and the greater curvature of the stomach. Two narrow clamps are then placed upon the stomach, from the upper and lower borders (Fig. 9). The clamp on the greater curvature must be placed in position first because the length of its blades corresponds to the length of the slot in the fenestrated clamp, namely 2⅜ inches. This length is that which has been found to make a suitable communication between the stomach and jejunum. The stomach beyond the clamps is removed by cautery. After a tiny opening is cut in the jejunum one blade of the fenestrated clamp is introduced into its lumen and the junction completed by the cautery technique already described. Figures 9, 10, and 11 show how the stitching is carried out and how the

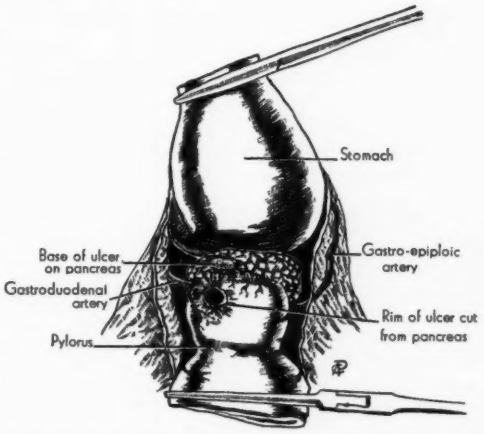


Fig. 5. In dissecting free the duodenum the base of a penetrating ulcer has been left on the pancreas.

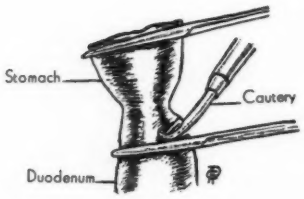


Fig. 6-

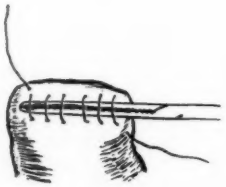

Fig. 7.

Fig. 8.

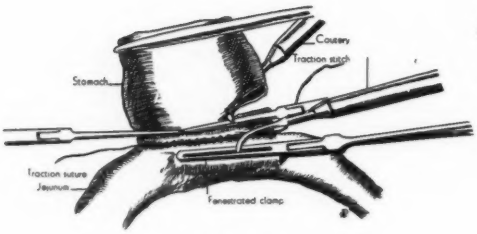


Fig. 9.

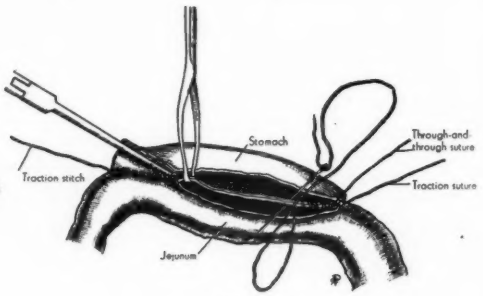


Fig. 10.

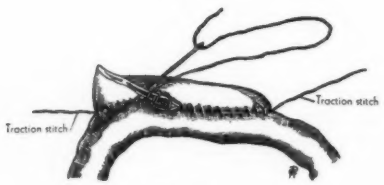


Fig. 11.

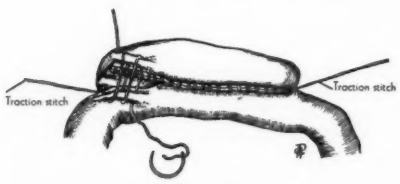


Fig. 12

Fig. 6. The affected segment of the duodenum is severed by cautery.

Fig. 7. First infolding seromuscular stitch.

Fig. 8. The closure is completed by invaginating the duodenum still farther the end of the same stitch being used.

Fig. 9. A sufficient length of stomach has been freed. The jejunum has been sutured to the stomach. The special clamps have been applied to the stomach and the fenestrated clamp to the jejunum. The stomach is being cut away with the cautery and the opening into the jejunum is being made with the same instrument.

Fig. 10. The posterior through-and-through suture.

Fig. 11. The anterior through-and-through suture has been inserted and is being carried on to close the redundant opening in the stomach.

Fig. 12. The through-and-through suture is being buried by a seromuscular stitch.

part of the stomach grasped by the clamp on the lesser curvature is closed. The method of inserting the seromuscular suture shown in Figure 12 is important. It will be noted that the unopened jejunum is sutured to the stomach distal to the anastomosis. If this is not done obstructive kinking might occur at the extremity of the communicating opening; for where this is, all the circularly running muscle fibers are divided so that a propulsive peristaltic wave cannot take place. But in the procedure described, any kinking at the lesser curvature is obliterated by the stream of chyme forced along by the intact jejunum between the anastomotic opening and the lesser curvature of the stomach. The abdomen is then closed.

### THE OPERATION FOR GASTRIC ULCER

The operation is carried out on the same principles. As the duodenum is cut across immediately distal to the pyloric sphincter it is not necessary to cut the pyloric part of the stomach across to get access for a difficult dissection. The difficulties in gastric resection are due to the ulcer penetrating into the pancreas and the great thick-

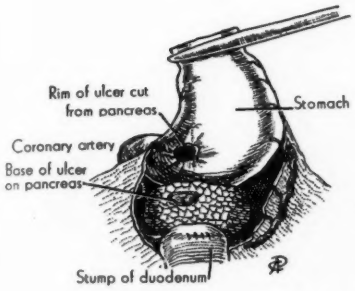


Fig. 13.

Fig. 13 left. Diagram to show how a posterior penetrating gastric ulcer is separated from the pancreas leaving its base untouched.

Fig. 14. Line of section of the stomach in the treatment of ulcers situated high in the viscus.

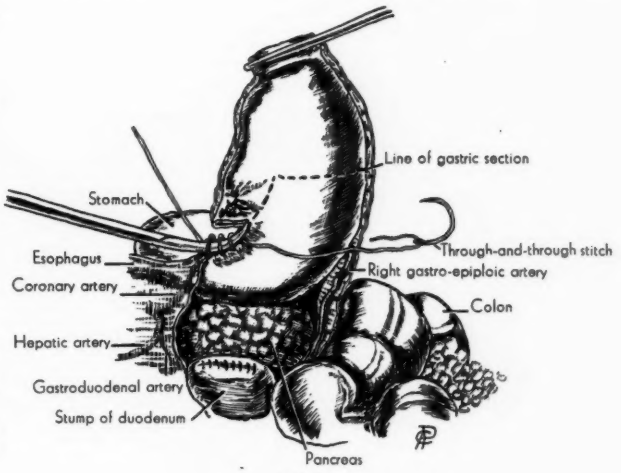


Fig. 14.

ening of the lesser omentum. In order to be able to resect, it must be possible, after opening into the lesser sac as described, to get the finger round the proximal boundary of the ulcer where it is adherent to the pancreas. The vessels on the greater curvature are ligated in the usual manner. On the lesser curvature great care must be taken. The tissue is very hard; often the individual vessels cannot be seen. Ligation should be done in small sections and always the thinnest catgut serviceable used because thin ligatures have less tendency to slip.

As soon as possible the stomach is turned over to the left. When the ulcer penetrates the pancreas its rim must be separated from this organ leaving the base untouched. This separation can usually be done avascularly by blunt force (Fig. 13). When the stomach has been sufficiently separated the clamps are applied to it proximal to the ulcer and the anastomosis effected as already described. Should the ulcer be so high that placing the clamps in this fashion would seem to lead to the sacrifice of an unnecessary amount of stomach, a gutter resection should be done. This is carried out as suggested in Figure 14. The clamp shown is a Moynihan cholecystectomy clamp. In these high ulcers the curved part of the incision in the stomach near the lesser curvature should be sewed up in two layers before the anastomosis is made with the straight part of the incision. Otherwise when the main bulk of the stomach has been cut off the upper part of the incision in the stomach recedes into the abdominal cavity and becomes very inaccessible.

Finally for very high ulcers on the posterior wall of the stomach near the esophagus another procedure may be adopted. The ulcer itself is excised with the cautery and the hole in the stomach is carefully sutured. The stomach is then anastomosed with the jejunum along a line running distal to the ulcer. I have never performed a complete gastrectomy for simple ulcer of the stomach and do not consider it justifiable.

During these operations the ordinary precautions against spreading infection, namely the isolation of the operative area by saline soaked packs, is adopted. The contents of the stomach and duodenum, as a rule, have very little infective power. However, one death already referred to took place from septic peritonitis, though the postmortem examination showed there had been no technical error, and the anastomosis was watertight.

After a trial of splanchnic combined with local, and with spinal anesthesia, these methods have been discarded as less satisfactory than general anesthesia with gas and ether. Recently a few patients have been operated upon under cyclopropane anesthesia. The results have been very promising. The patients do not have any solid food until the eighth day. Six weeks after leaving the hospital they are allowed to eat any food but warned that, for a year, their meals must be small, though they may be frequent.

# RADICAL TREATMENT OF INTRACTABLE PRURITUS ANI

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CERTAIN cases of intractable pruritus ani resist all the commonly employed forms of therapeusis. These cases usually have run the gamut of salves, lotions, injections, x-ray treatments, fulguration, and anything that any physician or any layman may have suggested. In desperation, patients so afflicted go from one physician to another, hoping against hope, that someone may have a remedy for this non-malignant but most incapacitating and nerve racking disease. At this stage, the perianal skin has undergone such advanced changes as to make it unrecognizable. Grossly and microscopically, malignant degeneration seems inevitable. These changes are due to the disease itself, to the constant scratching and irritation and to the various forms of therapy employed, particularly the sclerosing injections and the x-ray treatments. *Radical curative therapy at this time is imperative.*

In the armamentarium of the proctologist, there is no rational therapy for this type of case. The injection of oil soluble local anesthetics or alcohol does not eradicate the disease except in the event of widespread necrosis. The Ball operation and its various modifications attempt to deprive the part of sensation but fail to eradicate the diseased areas, and give but temporary relief.

The operation which we employed and which we will describe was used without knowledge of any like procedure having been performed before. Because of the good results obtained as evidenced in our follow-up, it was decided to report these cases. A search of the literature at this time revealed that somewhat similar procedures had been done about 40 years ago.

In 1898 Mathews (2) reported to the Louisville Surgical Society a case of severe pruritus ani treated by removal of the perianal skin. He excised the perianal skin and allowed the wound to heal by granulation. Later in his text book (3) he reported a somewhat different technique. After dissecting the skin free from the subcutaneous tissue he left it attached to the mucous membrane, split it into three sections, and tied ligatures at the base of each. These were left to slough off, and the wound was allowed to granulate.

From the Rectal Clinic and Surgical Service of Dr. Ralph Colp, Mt. Sinai Hospital, New York City.

In 1901 Hamilton independently described a somewhat similar operation. He made a circular incision at about the mucocutaneous junction, then an encompassing elliptical incision beyond the diseased area. The entire perianal skin between the two incisions was excised. The remaining skin was mobilized and then brought together anteriorly and posteriorly to the anus by several sutures. The mucous membrane was then sutured to the skin margins.

Montague states that surgeons did not take kindly to these methods because of the prolonged convalescence, severe postoperative pain, and the possible interference with sphincteric control. Apparently because of the fear of these distressing features the operation fell into disrepute and little has been heard about this method of treatment since.

When recently we were confronted with 3 cases of severe, intractable pruritus ani, accompanied by advanced skin changes, we decided to attempt a complete excision of all the diseased tissues. The technique of the operation which we devised for this purpose is as follows: The sphincter is gently dilated, *not divulsed*. The entire affected area is circumscribed by a circular incision which usually is at a radial distance of 2 to 3 inches from the anal margin (Fig. 1). The incision is carried down through the entire thickness of the skin until normal subcutaneous fat is encountered. The dissection is then continued in the subcutaneous tissue up to the mucocutaneous junction. In so doing, the fibers of the corrugator cutis ani muscles are severed, and the subcutaneous portion of the external sphincter muscle is carefully exposed and protected (Fig. 2). The cone shaped area of the skin is then split anteriorly and posteriorly (Fig. 3) in order to facilitate the subsequent section at the mucocutaneous junction, which is performed after clamps are placed on the mucous membrane to prevent retraction (Fig. 4). The gluteal skin is mobilized for a distance of 1 to 2 inches. All bleeding points are carefully controlled. Four silk mattress sutures are placed between the mucous membrane and the skin edges at equidistant points anteriorly, posteriorly, and laterally. Interrupted silk sutures are used to complete the approximation (Fig. 5). A small piece of vaseline gauze is in-

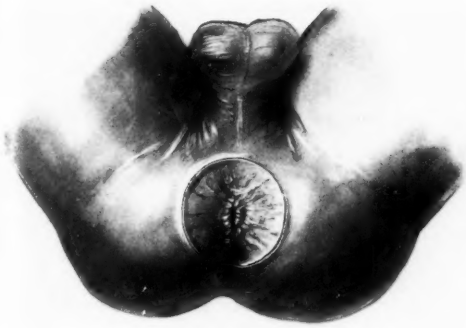

Fig. 1. Line of incision.

Fig. 2. Method of dissection.

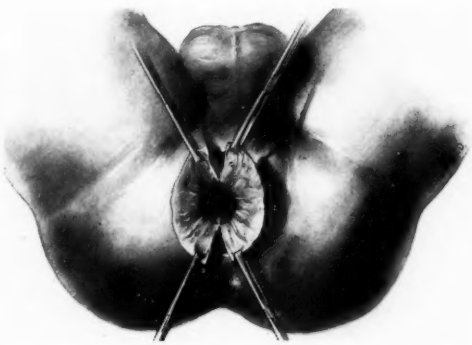

Fig. 3. Splitting of the cone-shaped area of skin.

Fig. 4. Clamps placed on mucous membrane to prevent retraction.

serted into the newly constructed anal canal, and the entire operative area is covered with vaseline gauze.

The results were most gratifying. Pruritus disappeared immediately after operation. Surprisingly little pain was present throughout the convalescence. The average period of hospitalization was only 11 days. The sutures, which were employed largely to prevent undue upward retraction of the mucous membrane and outward retraction of the skin, usually cut through between the fifth and eighth day, and moderate separation of the skin and mucous membrane occurred. This allowed the mucosa to retract to a level approximating the previous mucocutaneous junction. The intervening space filled in with granulation tissue and subsequently epithelialized. Complete healing occurred in from 6 to 10 weeks

Fig. 5. Interrupted silk sutures complete approximation.

after operation. Stricture was not encountered. In 1 case there was a slight narrowing which promptly responded to two digital dilatations. Temporary incontinence was observed in 2 of the 3 cases. This lasted less than 2 weeks and good sphincteric tone gradually returned, as could be demonstrated by marked contraction of the sphincter upon irritation of the new perianal skin.

In addition to the fact that the patients were overjoyed with the complete cure effected by this procedure, their general condition improved remarkably.

### CASE REPORTS

CASE 1. H.F. (Hospital No. 391427), a 63 year old man, was admitted to the rectal clinic, December, 1935, complaining of intense itching in and about the anus for the past 15 years. During this period he had at first tried many conservative methods of treatment, but finally in May, 1932, he was admitted to another hospital where an anal operation was performed. Communication with this hospital revealed that the operation consisted of "excision of redundant anal tissue." Relief from the pruritus was very short lived and after again attempting various forms of non-operative therapy he was re-admitted to the same hospital in May, 1935. This time a hemorrhoidectomy was performed. The symptoms became so severe shortly after this that the patient could not sleep, was in constant pain, and began to lose weight very rapidly.

Physical examination disclosed an elderly male in apparent discomfort. General examination was essentially negative. There was no evidence of fungus infection of the feet.

Proctologic inspection disclosed a cauliflower-like growth surrounding the entire anus and extending outward for a distance of 2 inches from the anal margin. The skin was hypertrophic, gray white in color, and markedly thickened and indurated. Numerous ulcerations were present, as well as polyp-like nodules. Beyond this area the skin was reddened, thickened, and excoriated. Palpation revealed marked anal spasm but otherwise the region was negative. Anoscopy showed the anal canal to be normal in appearance. Small internal hemorrhoids were noted but no hypertrophied papillæ or enlarged crypts. Sigmoidoscopy with the instrument passed full length, revealed the mucosa normal in appearance throughout.

The stool was brown in color and of normal consistency, reaction acid, and no blood, ova, or parasites were found.

The blood Wassermann was negative, the blood sugar was 100 milligrams per 100 cubic centimeters, the blood urea was 10 milligrams per 100 cubic centimeters. Hemoglobin was 82 per cent; white blood cells 6800, of which 65 per cent were polynuclears, 29 per cent lymphocytes, 3 per cent monocytes and 3 per cent eosinophiles. The urine was negative.

Biopsy of the perianal skin was performed. The pathological report was "fibro-epithelial polyp with conspicuous acanthosis and inflammation. *Please watch patient.*" The patient was then referred to the radiotherapy department for consultation. The opinion rendered was "Radiation is not indicated in a condition of this kind. Advise wide surgical removal because this is undoubtedly a precancerous condition."

In March, 1936, the patient was admitted to the surgical service for radical operation, which was performed as described above by Dr. Ralph Colp.

Pathologic report—macroscopic examination: "Specimen consists of a resected anus and perianal tissue. The skin and subcutaneous tissue about the anus is roughly oval in shape and measures approximately 8.5 by 6 centimeters. The perianal skin appears thickened, coarse, with several irregular folds which are quite firm. Here and there are small irregular ulcerations approximately 3 by 2 millimeters in diameter. Partially encircling the anus, a short distance from it, is a shallow ulcerated area, the base of which is covered by granulation tissue. It measures 3 centimeters by ½ centimeter. No unusual features are noted in the subcutaneous tissue."

Microscopic examination was reported: "Acanthosis and chronic non-specific inflammation with ulceration. No evidence of tumor."

The postoperative course was uneventful. About the sixth day the sutures cut through and were removed. Treatment then consisted of frequent hot baths and wet dressings. The patient was discharged on the ninth day and further dressings were done at the clinic. The wound healed by granulation and was completely epithelialized in 5 weeks. For the first 2 weeks there was some incontinence of liquid feces but as healing progressed he regained complete control. He was last seen on January 5, 1938, at which time he reported that he had been completely free from pruritus since operation 22 months before. The new perianal skin appeared soft and normal in texture. Control was perfect and there was no stricture. There was however a slight eversion of mucosa on one side. Digital examination revealed good sphincteric tone. The general physical examination, however, was even more satisfactory. His entire appearance was altered. He had been transformed from a harassed, gaunt, chronically sick old man into a cheerful, pleasant personality.

The indications for radical surgical therapy in this case were very definite; 15 years of severe itching and pain, a skin lesion that was becoming progressively worse, showing no tendency at any time to regress, a pathological report that implied danger of malignancy and a clinical appearance that caused both the surgeon and the radiotherapist to feel certain of its malignant potentialities.

CASE 2. H.I. (Hospital No. 398976), a 28 year old German chef, was admitted to the rectal clinic on September 18, 1936, complaining of itching, pain, and lumps about the anus. His illness had started 10 months prior to admission with the appearance of several small warty growths in the perianal region. These had increased rapidly in size and number, and were associated with severe pain and intense itching. He had been treated in two clinics where fulguration and partial excision had been performed, but the relief had been of very short duration. After a few weeks the growths recurred and grew rapidly again. He then received several x-ray treatments with but slight improvement. The patient had noticed that after the temporary improvement which followed the fulguration, excision, and x-ray treatments, the growths tended to recur with greater rapidity and the sumptoms of pain and itching became more intense. His symptoms became so severe that he was unable to sleep, he could not continue his occupation, his appetite failed, and he lost 20 pounds in weight. Several weeks before admission he observed the appearance of a number of growths on the shaft and glans of the penis.

Inquiry into his past history disclosed a traumatic

hemothorax 12 years ago and pneumonia 7 years ago. Shortly after he recovered from the pneumonia, he became infected with gonorrhea and syphilis. The gonorrheal infection cleared up rapidly; the luetic infection had been treated with bismuth and intravenous arsenicals. The exact number of injections was not known. The last treatment had been given 1 year ago. Blood and spinal fluid Wassermann tests done during the past year were negative.

General physical examination was negative except for the local condition.

Proctological examination: The entire perianal area for a distance of 2 to 3 inches from the mucocutaneous junction was covered with numerous large and small condylomas. A few of these appeared roughened and hornified. The skin was quite red, markedly thickened, indurated, and tender. There were innumerable scratch marks.

Digital examination: In the anal canal were felt a number of small hard warty growths similar to those seen on the perianal skin.

Anoscopy revealed the rectal mucosa to be normal in appearance; there was no evidence of proctitis or infected crypts. In the anal canal, distal to the mucocutaneous junction, were seen several condylomas, similar in appearance but smaller in size than those on the perianal skin.

Sigmoidoscopy with the instrument passed full length showed no abnormality.

Examination of the genitalia revealed several small condylomas on the glans and shaft of the penis.

The blood Wassermann was negative. Rectal smears for gonorrhea were negative. The Frei test was negative. The urine was negative. One of the condylomas was removed and pathological sections were made. The report verified the clinical diagnosis of condyloma.

The patient was desperate and begged for any method of treatment, however radical, which would give immediate and lasting relief. It was felt that conservative methods had been unsuccessful and that radical surgery was indicated. Accordingly, the patient was admitted to the surgical service and was operated upon on September 30, 1936 by Dr. Amiel Glass.

After operation, the patient did very well. Most of the sutures cut through on the sixth and seventh days and separation occurred between the skin and the mucous membrane. This area rapidly became filled with healthy granulation tissue. He was discharged on the fifteenth day after operation. Curiously enough, at the time he left the hospital, it was noticed that the condylomas of the penis had completely disappeared, although they had received no local treatment.

Six weeks after operation, the wound was completely healed, and the patient felt well. Bowel movements were normal and sphincter control was perfect.

The patient was last seen in January, 1937, at which time the anus appeared normal except for slight narrowing of the anal orifice. This required only two digital dilatations. Shortly after this, the patient moved to another city because employment was available there. He was last heard from about one year after operation when he communicated with his family physician and informed him that he was entirely symptom free and normal in every respect.

The indications for radical surgical therapy in this case were very definite: continuous intense pruritus and severe pain for 10 months, quick recurrence of the lesions after surgical removal and electrodessication, and failure to obtain any relief by x-ray or any other form of treatment.

Case 3. L.R. (Hospital No. 414601), a 45 year old physician, was first seen by one of us (S.D.M.) in May, 1937. His history dated back to 1925 at which time he had a few small perianal warts treated by fulguration. As a result of this procedure he sustained burns of the perianal skin which healed very slowly. Pruritus first set in at this time. The itching became progressively worse and was associated with painful fissures. Many forms of local palliative therapy were employed without relief. In 1928, after suffering intensely for 3 years, he was seen by a dermatologist who instituted x-ray therapy. The patient secured moderate relief for about 2 months, but then relapsed and the pruritus and pain became worse than ever. During the next several years he was treated by many physicians and many types of treatment were used. In 1932, he had a series of injections of oil soluble anesthetics. Multiple abscesses developed following this, and new ones continued to appear during the next 6 months. X-ray treatments were again given, but this time failed to afford even temporary relief. The patient could not sleep without the use of large doses of sedatives and even then spent many sleepless nights. He was constantly in misery, his general health was rapidly failing, and he could not attend to his work. His mental state was such that he was prepared to undergo any procedure rather than continue life as it was.

General physical examination was essentially negative.

Proctologic examination: The perianal skin for a distance of 2 inches from the anal margins appeared markedly thickened and hypertrophic, and was thrown into edematous folds. Between these folds were deep ragged infected fissures. The skin immediately adjacent to the anal margins appeared "parboiled" and felt quite hard, whereas the more distant skin appeared erythematous and felt indurated.

Digital examination: The ampulla of the rectum and the upper end of the anal canal were normal. The distal portion of the anal canal was tender and indurated.

Anoscopy revealed a few small internal hemorrhoids. No crypts or hypertrophied papillæ were found. The pectinate line was completely obliterated by the pathological process which extended up to it.

Sigmoidoscopy with instrument passed full length revealed no abnormalities. Skin scrapings were negative for fungi. Urinalysis was negative.

During the ensuing 4 months the patient was treated with bland ointments of many varieties and with an autogenous vaccine made from one of the many perianal abscesses which he developed during this period. The condition became progressively worse, both subjectively and objectively, and in September, 1937, he agreed, in desperation, to submit to radical surgery. He was admitted to the Private Pavilion of the Mt. Sinai Hospital, and operated upon on September 27, 1937, by one of us (S.D.M.). The procedure described was employed.

Pathologic report: "Specimen consists of two portions of skin of similar size measuring 8 by 4.5 centimeters in the greatest diameter. The epithelial surface is somewhat puckered in areas and also shows an irregular yellowish pink firm thickening. The subcutaneous tissue is slightly hemorrhagic."

Microscopic examination report was: "Hyperkeratosis with chronic inflammation."

The postoperative course was remarkably smooth. There was practically no pain until the fifth day when the sutures cut through. Upon removal of the sutures, this slight discomfort disappeared. Healthy granulations appeared where the skin and mucous membrane had separated. The patient was discharged on the eighth postoperative day and started to resume practice on the tenth day. During the first 2 weeks after operation, sphincteric control was not perfect, but as healing progressed, normal sphincter function returned. The wound was completely healed 5 weeks after operation. The new skin appeared normal, sphincter tone was good, there was no narrowing of the anal orifice, and the patient was completely free from pruritus and pain.

He was last seen 4 months after operation. He had gained 20 pounds, was working harder than ever, and had been sleeping well without the use of sedatives. There were no rectal complaints.

The indications for radical surgical therapy in this case were also very definite: A 12 year history of intractable pruritus and pain, an intolerable existence, failure to obtain relief for even short periods with any form of therapy, and a clinical appearance that gave the impression of possible malignant degeneration.

## SUMMARY

1. Three cases of long standing, severe pruritus ani with extreme skin changes have been presented.

All 3 patients had been treated by the usual accepted methods with no lasting relief.

2. A radical operation which has been utilized in an attempt to cure these patients permanently has been described.

3. Results have been uniformly satisfactory, all three patients having been completely relieved of their pruritus for the first time since its onset. They have been followed and have remained well for periods varying from 4 to 22 months after operation.

4. Contrary to general belief, this type of operation is not followed by long convalescence, severe postoperative pain, stricture, or sphincteric incontinence.

5. This method is not advocated for the treatment of the usual case of pruritus ani. It is intended only for cases of long standing, intractable pruritus ani, particularly those with pronounced and possibly premalignant skin changes.

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# CARCINOMA OF THE MAJOR VESTIBULAR (BARTHOLIN) GLAND

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MALIGNANT disease of Bartholin's or the major vestibular gland, is rare and not often considered in the differential diagnosis of diseases of that organ. Usually the diagnosis is made only at operation when the pre-operative diagnosis of cyst or abscess is upset by the finding of a solid neoplasm. Here, as elsewhere in the body, careful observation of the patient and timely surgical intervention, together with the laboratory examination of the excised material, offer the only hope of recovery.

## EMBRYOLOGY AND NORMAL ANATOMY OF BARTHOLIN'S GLAND

Huguier was the first to describe extensively the gross anatomy of the major vestibular gland. The microscopic picture, begun by Langerhans, was not elaborated until the present century when Jambon and Chaboux, as well as Cullen in this country, carefully studied series of specimens from various age groups. Schroeder's résumé of the embryology and anatomy is the most concise yet published, and completes the material upon which this study is based.

The major vestibular gland is the homologue of the bulbo-urethral gland of Cowper in the male but develops later. Schaffer, in his textbook, refers the reader to the description of Cowper's gland in discussing Bartholin's gland. The unequal development of the glands, beginning about the third fetal month, as soon as there is external sex differentiation, starts in the vestibular epithelium of the urogenital sinus. The latter is very definitely developed in the embryo at the end of the fourth month. Differentiation is virtually complete by birth. At birth, tubules of a single layer of pale cuboidal cells, staining with muci-carmine, lie in an interstitium, at the periphery of which are other tubules of darker smaller muci-carmine-negative cells.

Smaller ducts are formed of multilayered epithelium, cuboidal for the most part, and cylindrical directly on the lumen. The excretory duct has, according to Schroeder, a multilayered epithelium similar to transitional epithelium with squamous cells basally and columnar superficially. The gland diminishes in size between 9 and 12 years and then definitely increases at puberty, to decrease and atrophy in senility.

The bilateral gland lies on the urogenital diaphragm surrounded by the vascular vestibular bulb, and is separated from the vagina by perineal soft tissues and the vaginal sphincter bundles. The gross measurements are given as 11 to 15 millimeters in length and 0.5–3.0 millimeters in width. In more than three-quarters of a series of cases the chief excretory ducts lie within 1 to 2 millimeters of the border of the labium minor, with a lumen of 1 millimeter and a length of 1 centimeter (Schroeder). This duct opens into the vestibule just below the outer border of the hymen.

Cullen compared the gland to an unevenly developed bunch of grapes, with the main duct compared to the stem, the secondary and terminal ducts or excretory canals to the branches, and the lobules to the fruit. In the adult, the pale columnar cells of the gland alveoli have small deep-staining nuclei and pale cytoplasm staining variably with muci-carmine. These cells rest on a membrana propria of the surrounding connective tissue which is highly vascular with much elastica and less smooth muscle. The gland terminal ducts or excretory canals, lined by one to several layers of cuboidal to columnar cells, unite in an ampulla lined by columnar epithelium, and here the true excretory or main duct begins. As it enlarges, the main duct is lined by flattened transitional or squamous epithelium and by vestibular squamous epithelium in its outer portion. The gland has been characterized by some as acinar and by others as tubular.

Outside the capsule are large calibered nutrient arteries and veins, nerves, and striated muscle bundles. The striated muscle within the capsule, however, is sparse and is found only about the large excretory canals or ducts. The blood supply, by large arterioles, is from the internal pudendal and bulbar arteries and is distributed by numerous capillaries between the acini, which end peripherally in venules emptying into the bulbar and pudendal sinuses accompanied by nerves and lymphatics. The secretion of mucus is intermittent because of neurogenic alteration.

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Fig. 1. Photomicrograph of structures from inflamed major vestibular (Bartholin) gland: *a*, normal acini communicating with the duct, *b*, completely obliterated by hyperplastic transitional epithelium (compare with Fig. 3 for similarity of cell type). Low power.

### DIAGNOSTIC CRITERIA OF PRIMARY CARCINOMA OF BARTHOLIN'S GLAND

It is not always possible to determine whether Bartholin's gland is the primary site of the malignant vulval neoplasm. The cases reported by Ratkowski and by Taussig are probably secondary in the gland, while Savournin's case is not one of carcinoma. Melanoma doubtless was the correct diagnosis in the case published by Sinn. These cases are not included in our summary.

Honan enumerated four criteria which must be satisfied before an indisputable diagnosis of primary carcinoma of Bartholin's gland is made. These are: (1) typical vulval site, (2) position deep in the labium, (3) connection with the gland duct, and (4) the presence of intact gland tissue. These conditions are satisfied in our first case. The typical site in the lower third of the labium majus and deep within it was noted in the second case. No normal portions of the gland or duct were found in the microscopic sections available and the gross specimen had long since been discarded (1926).

If, in addition to the requirements already mentioned, the skin is intact and the carcinoma a frank adenocarcinoma, as suggested by Schaeffer, the major vestibular gland is unquestionably the primary site. Numerous authors point out that the smaller ducts communicating with the acini (Fig. 1, *a*) are lined by transitional epithelium (Fig. 1, *b*), a fact confirmed by us in sections of routine surgical material. This type of epithelium is not present in vulval structures other than Bartholin's gland. A study of many sections of the second case and a comparison with the normal disclose a striking similarity to the cell types of the normal transitional epithelium and justify classing it among the carcinomas of the major vestibular gland.

The problem of the primary site is not as readily clarified in cases of squamous cell carcinoma. If normal glandular structures lined by columnar epithelium are seen contiguous to, or continuous with, the squamous cell carcinoma, it has been assumed that the latter arose from metaplastic epithelium. Frank and Kehrer speak of such a possibility, although such metaplasia has not been encountered in routine surgical material examined by us. A more likely point of origin is the stratified squamous epithelium which normally extends inward from the skin and lines the ducts for a variable distance. That distance may vary with the extent of destruction of the original transitional epithelium and its replacement by the squamous epithelium. Unless the squamous cell carcinoma is seen to begin in the skin over the major vestibular glands, squamous cell carcinoma of this area involving such glandular structures must be included among the carcinomas of Bartholin's gland.

### ANALYSIS OF LITERATURE

*Frequency.* Bartholin's gland carcinoma is relatively rare. Burghele could report only 3 primary vulval carcinomas among 20,000 patients under his care, one of which was of the major vestibular gland. At the Mayo Clinic, according to Hunt and Powell, there were 170 operations on the major vestibular gland over a period of 15 years. Of these, 150 operations were for cysts, 17 for infections, and 2 for carcinoma. Over a later 10 year period, Mayo and Barber reported 281 cysts, of which 154 were operated upon. Three of these were carcinomas. Lynch wrote that "this tumor is undoubtedly rare, yet the literature does not properly express the frequency, since vulval growths are usually seen by men in general practice who do not report them and who may be content merely with their removal."

At the New York Post-Graduate Hospital during the past two decades there are recorded, among the lesions of Bartholin's gland, 14 infections, 70 abscesses, 92 cysts, and the 2 carcinomas here reported, a frequency of 1.1 per cent. These figures are generally similar to those of the Mayo Clinic where the carcinomas of Bartholin's gland comprised 0.8 to 0.9 per cent of all diseases of that organ. There have now been recorded or pub-

lished a total of 56 such cases, not including the two described in this paper.

*Type of carcinoma.* In 7 instances no information is given as to the type of carcinoma. Two cases are described only as "anaplastic," and one each as solid, small cell and adenosquamous cell carcinoma. Adenocarcinomas numbered 29, and squamous cell carcinomas, 12.

*Age.* The youngest patient is that of Beckmann, 19 years. Four cases are reported of patients between 26 and 30 years of age. Seven cases are recorded in the fourth decade, 16 in the fifth, 12 in the sixth, 5 in the seventh, and 3 in the eighth decade of life; the decades before and after the menopause are the periods of life most affected.

*Symptoms.* The most frequent symptom is the appearance of a mass in either side of the vulva, as described in 35 cases, and present for an average of 15 months before medical aid was sought. Whether a benign condition was at first present cannot be answered. Pain was a much less prominent symptom. Dyspareunia and discomfort on walking and sitting were noted. In 13 of 37 cases the inguinal nodes were involved when the patients were first examined, although Kehrer and Tobler both state that the regional lymph nodes are involved relatively late in the disease. Femoral and iliac node involvement has also been recorded. Metastases have been found in vertebræ (Fabricius), pelvis and chest (Lyle).

*Therapy.* In most instances resection was performed, although in several cases incision was made before the neoplastic character of the condition was determined. Radiation therapy, both x-ray and radium, was also used, usually after operation as in Hunt and Powell's third case which was well after 2 recurrences, and in Healy's first case, each 11 years after the original operation. Without radiotherapy the longest period of life after operation (the patient still alive at the time of publication) was 3½ years (Eden, Polaillon). Fabricius' patient died 5 years after resection. Recurrence took place after a few months in most cases and a second recurrence was frequent. Most authors do not record the date of death or necropsy findings.

## CASE HISTORIES

CASE 1. R. A. R. (a patient of Dr. Eugene L. Jewett), 32 years old, white, was admitted to the New York Post-Graduate Hospital August 28, 1934, for x-ray therapy. In 1926, she is said to have been treated for pulmonary tuberculosis. About 3 months before admission she noticed a small mass in the left half of the vulva which caused little or no pain. The mass grew larger and became distinctly painful. Physicians consulted made a diagnosis of an infected Bartholin's gland but did not advise operation. Because of the rapid increase in size and the patient's insistence, the mass was removed and submitted for examination.

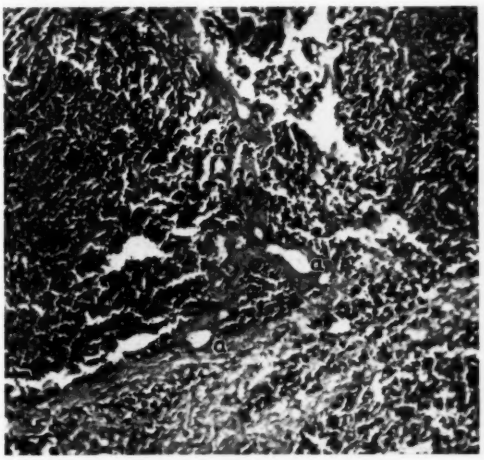


Fig. 2. Photomicrograph of anaplastic adenocarcinoma of the major vestibular (Bartholin) gland (Case 1): *a*, Normal ducts lined by cuboidal to columnar epithelium. Low power.

At admission to hospital there were no signs of acute or chronic disease except for slight discrepancy in size of pupils. The wound in the labium majus was partly healed. The left inguinal nodes were enlarged, firm, and movable. The local site, the vulva, both groins and the lower abdomen were radiated separately. The patient was discharged improved, August 30, 1934.

She was readmitted 3½ months later for 3 days. Amenorrhea followed the x-ray therapy of August. In November, 1934, small nodes were excised from the left groin but not submitted for examination. Because of the recurrence in the groin where, beneath the recent surgical scar, several soft matted glands were palpated, excision and further x-ray radiation were advised. The complement fixation test on spinal fluid for syphilis was positive, with a colloidal gold reaction of 1233310000.

At operation, December 14, 1937, two deep inguinal nodes, matted together and apparently well encapsulated, were removed. There were no signs of recurrence at the site of the primary neoplasm of Bartholin's gland. Following discharge from the hospital, x-ray radiation was applied to the abdomen and perineum, to the neck and chest (February and March, 1935) and once to the groin (May, 1935). Antiluetic treatment was given at the same time.

In May, 1935, the patient, who had been suffering from dizziness, weakness, nausea, and vomiting, was knocked down by a taxicab. She was unconscious for about 10 minutes and was removed to another hospital where she remained until transferred to the New York Post-Graduate Hospital the following month. Following the accident all symptoms became aggravated and headache, previously unnoted, now appeared. There were also periods of apparent confusion and failure to respond normally to questions.

At admission the mental status was good although the patient complained of marked deafness. She was emaciated and anemic. The pupils were irregular and did not respond to light. A large, hard, circumscribed mass, pal-

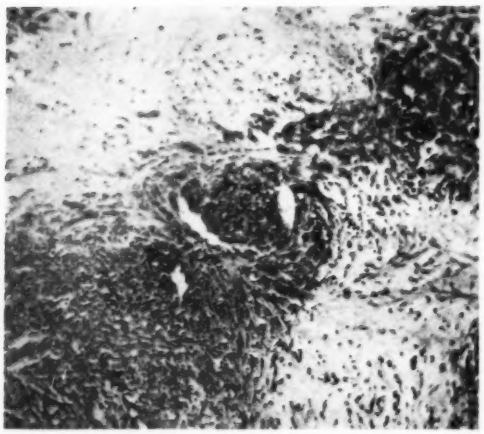

Fig. 3. Photomicrograph of transitional cell carcinoma of the major vestibular (Bartholin) gland (Case 2). Low power. (Compare with Figure 1 for similarity of cell type.)

pated in the epigastrium, extended into the right upper quadrant. There were scars on the anterior abdominal wall, possibly the result of the radiation, and the pubic hair was absent. In the left inguinal region, in addition to the operation scars, there were a few small enlarged nodes. There was no sign of recurrence at the primary site, and a purulent vaginal discharge was present.

Otological examination attributed the deafness to the general condition, aggravated by the trauma of injury. Vomiting continued and the abdominal mass increased in size. Incontinence of urine and feces appeared, accompanied by mental stupor and drowsiness. The temperature slowly rose to 105 to 106 degrees, at the time of death.

At necropsy, 13 hours later, there was no evidence of recurrence of the neoplasm at the original site of the left labium majus. Metastases were found in the brain, dura, lungs, heart, liver, pancreas, adrenals, kidneys, left ovary, and mediastinal lymph nodes. These were more or less sharply delimited from the surrounding tissues and were formed of moderately soft, gray to grayish white areas with central zones of necrosis and hemorrhage.

The dural metastases were situated on the left posterior portion of the falx cerebri, excavating the adjacent areas of the left occipital lobe. A marked dilatation of the lateral ventricles and particularly of the third ventricle, was caused by a 3 centimeter metastasis resting on the anterior portion of the fourth ventricle floor and slightly adherent to the culmen. The cardiac metastasis was in the endocardial tissues.

*Description of specimen.* The primary neoplasm removed from the left labium majus, 30 by 22 by 20 millimeters was covered by a thin connective tissue capsule. On section, firm gray tissue intermingled with yellow tissue was indefinitely lobulated. Microscopically there was a diffuse proliferation of deep staining closely-packed small cells arranged in minute clusters or thin strands separated by a small amount of vascular connective tissue (Fig. 2). The neoplastic cells were generally polyhedral with scanty cytoplasm and dense pycnotic nuclei. Some cells were fusiform and where the gland structures were well formed they were cuboidal. Mitotic division figures were not numerous. In the neoplasm there were cross sections of small gland ducts lined by normal cuboidal to columnar (Fig. 2,*a*) epithelium, while others were filled with exfoliated epithelium similar to that of the main tumor mass. In scattered glandular structures both neoplastic and normal cuboidal epithelium lined the acini. The diagnosis was "anaplastic adenocarcinoma."

The histological picture seen in the inguinal node first removed, and in the metastases at necropsy was generally similar to that of the primary labial growth. In the pia the neoplastic cells formed large distended blood sinuses.

CASE 2. A. J. G., 53 years old (a patient of Dr. John H. Morris), white, single, was in the New York Post-Graduate Hospital for a fortnight in May, 1926. She complained of "a large swelling in the vagina." About 4 months before, she had noted a swelling in the left side of the vulva which gradually increased in size. During the few weeks before admission there was marked pain, as well as difficulty in sitting and on defecation. A provisional diagnosis of "Bartholin cyst" was made on finding a tense, non-tender large swelling between vagina and rectum. At operation, resection, a solid tumor was found. There were rapidly growing pelvic metastases and the patient died less than 6 months later.

The specimen submitted for examination consisted of 150 cubic centimeters of broken-down tissue, including moderately soft tissue and large blood clots. Microscopically there was a connective tissue stroma arranged, for the most part, as slender papillæ, on which were grouped thick layers of epithelial cells, irregular in shape with numerous mitotic figures. The cells resembled those of transitional epithelium (Fig. 3). The diagnosis was "papillary carcinoma, apparently derived from transitional epithelium."

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# A TECHNIQUE FOR TRANSFUSION OF BLOOD INTO THE ABDOMINAL AORTA

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THE administration of blood to infants and young children frequently presents difficulties. Usually, the superficial veins of the arms, hands, and feet are small and will not permit the use of a very large caliber needle for venipuncture. Whereas the anterior fontanel of infants may be used as the site of injection, it does present certain hazards; namely, the danger of extradural leakage of blood and the possibility of infection. The procedure of cutting down on the veins of children may be used, but parents occasionally object to this because unsightly scars may result. Then too, if the physician only occasionally performs transfusion by cutting down on the vein, there is a strong possibility that the vein may be injured and that an unsuccessful attempt to administer blood may result.

Aortic puncture has been performed previously[1] on adults for the purpose of making arteriographic studies of the abdominal and pelvic organs and of the lower extremities, by injecting into the abdominal aorta an opaque substance which may be visualized roentgenologically. In addition, various aniline dyes have been injected into the abdominal aorta for the purpose of combating peritonitis. It was felt that by this procedure a greater concentration of antiseptic solution could be brought in contact with the region involved than could be accomplished if the aniline dye were injected intravenously. It occurred to me that if the procedure were simple enough, the administration of blood to infants and young children might be satisfactorily accomplished by means of puncture of the abdominal aorta.

## TECHNIQUE OF AORTIC PUNCTURE

The patient is placed in the ventral decubitus position or he may lie on the left side with the head flexed toward the thorax and both knees brought up toward the abdomen (Fig. 1). The site of injection is usually at the level of the twelfth thoracic vertebra, but it may vary from the tenth thoracic vertebra to the second lumbar vertebra. The midline of the vertebra is palpated and a cutaneous wheal is raised with a 0.5 per cent solution of procaine or of metycaine, at the level of the twelfth thoracic vertebra, just below the twelfth rib and at a point approximately 5 centimeters from the midline. In cases in which the patients are newborn infants or infants less than 3 months of age, a special needle of No. 21 gauge and about 50 millimeters in length which has a bayonet lock is introduced through the cutaneous wheal toward the body of the vertebra. When the body of the vertebra has been contacted the needle is withdrawn slightly and introduced deeper into the tissue tangentially with the body of the twelfth thoracic vertebra (Fig. 1). As the point of the needle approaches the anterior portion of the body of the vertebra, it is very close to the abdominal aorta, and by advancing the needle ¼ to ½ inch (0.64 to 1.27 cm.) further, a throbbing sensation may be felt in the needle, which will indicate that the aorta is in close proximity and that a slight thrust anteriorly will cause the needle to enter the abdominal aorta. It should be pointed out that the injection should not be attempted from the right side inasmuch as one would contact the vena cava which is not as suitable a site for injection as is the abdominal aorta. When successful puncture of the aorta has been accomplished, bright red blood will drip from the end of the needle; as the caliber of the needle is small there will not be any gush of blood from the lumen of the needle. In addition, the systolic pressure in this portion of the abdominal aorta is about 50 to 80 millimeters of mercury; in other words, it is equivalent to the average diastolic pressure in the arm. As there is a definite pressure to overcome, I have found it most satisfactory to administer the blood by means of a syringe which is fastened securely by means of the bayonet lock. The citrate method of transfusion is used because of its simplicity. The amount of blood administered by this method will vary according to each individual case, but as much as 200 cubic centimeters may be given easily and within a half hour. I have had the opportunity to inspect the abdominal aorta in 2 cases in which death resulted from systemic dis-

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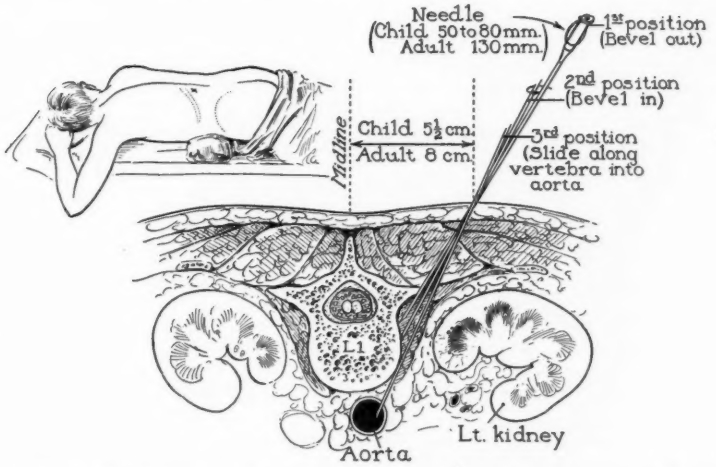


Fig. 1. The position of the patient and the various steps in puncture of the abdominal aorta.

ease and in both instances the puncture in the aorta was found and there was no evidence of undue trauma or evidence of leakage about the site of injection. As there are more highly developed muscular coats in the wall of the aorta than there are in the vena cava, it is safer from the standpoint of leakage to use the aorta as a site of injection rather than the vena cava. Furthermore, the vena cava is slightly more inaccessible than the aorta as it would have to be approached from the right side of the vertebral column and in many instances it lies practically in front of the body of the vertebra, whereas the aorta is usually placed more toward the left lateral margin of the body of the vertebra.

This is a new technique for the transfusion of blood into the abdominal aorta. The method has been used to transfuse blood into 6 infants and the results have been satisfactory. In one case, in which the patient was an adult, the method of treatment was used for arteriography with contrast media.

The technique is somewhat more simple if the patients are children than it is if they are adults, inasmuch as the muscular and skeletal development of children is not so marked. The method is not to be advocated in the hands of persons who do only an occasional transfusion, but is offered as an adjunct for those who are doing transfusions regularly.

# CARCINOMA OF THE RECTUM AND RECTOSIGMOID LIGATION OF THE INTERNAL ILIAC ARTERIES

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HEMOSTASIS in operations for cancer of the rectum and rectosigmoid is a problem which in times past has prolonged the time of operation in some cases. The patient suffers not only from prolongation of operating time but from loss of blood. Hence, any method which will render the problem of hemostasis easier is worthy of consideration. With this in mind the suggestion was made that preceding the removal of cancer from this region the internal iliac arteries be ligated. Especial stress is laid on this point, as, while neither new nor original, it is not done as a routine measure for cancer of the rectum and rectosigmoid.

Ligating the internal iliac arteries seems so radical that one is impelled to consult a textbook on anatomy to refresh his mind and to recall that in the pelvis among the arteries which come from the internal iliacs are the vesical, uterine, inferior hemorrhoidal, and the obturator. Collateral circulation supplies the necessary blood to other pelvic organs when the internal iliac arteries are ligated.

The literature has been searched to see if the procedure of ligation of the internal iliacs has been recommended or practiced elsewhere. Our search proves the old contention that there is little that has not been tried before. The oldest reference discovered by the writer is noted as follows: Damon B. Pfeiffer, in the "Annual Oration in Surgery" before the Philadelphia Academy of Surgery, March 7, 1927, stated:

"Quénu, in October, 1896, operated successfully upon a woman of 50 years, and separated the perineal from the abdominal stage of the operation by an interval of 6 days. This is the first instance of the two stage operation which has of late years been extensively employed in various forms. He did much to standardize and popularize the combined operation and it is generally known by his name. He emphasized the importance of asepsis and good hemostasis. He advised preliminary ligation of the internal iliac arteries, but this step has now been abandoned in most quarters as unnecessary."[1]

A later reference is in Ravdin's translation of Kirschner's *Operative Surgery*, in the 1933 edition, page 444, in which he states:

"If amputation of the rectum has been decided upon as a single stage operation, it is advisable, as the first operative measure, to ligate both internal iliac arteries, since this greatly lessens the bleeding during the freeing of the rectum from the pelvic wall. However, if a resection is planned, the ligation of these two vessels should be omitted, since they give rise to the middle and inferior hemorrhoidal arteries supplying the rectum."

The latest reference the writer has found to ligation of the internal arteries in cancer surgery, is the case operated upon October 27, 1937, in the Michael Reese Hospital, Chicago, during the Clinical Congress of the American College of Surgeons, by Dr. William Rubovits. In a personal communication he states:

"Bilateral ligation of iliacs has been done in Michael Reese Hospital, only for the relief of uncontrollable uterine bleeding in carcinoma. I have done the operation 3 times. The total number in our department is 6. When the vessels have been divided between ligatures (5 cases), bleeding has stopped immediately. The patient's condition improves rapidly. My hope is that the procedure may prove valuable when done extraperitoneally, if possible, or transperitoneally, when the problem presents itself in the course of a uterine operation accompanied by almost uncontrollable bleeding. This has occurred twice in my experience when fibroids invaded the retroperitoneal space."

So it is shown that the procedure practiced by Quénu in 1896 and by other able surgeons, then abandoned as unnecessary, is again finding a useful application. It would be extremely helpful in cases in which the perineum, anus, levator ani muscles, and related structures are to be removed.

A piece of original research on the collateral circulation of the pelvis and perineum was carried out as the result of the outcome of my recent case, in which the internal iliac arteries were ligated for cancer of the rectum and rectosigmoid. Dr. Arthur M. Wright, professor of surgery, and Dr. Benjamin G. P. Shafiroff, of the department of experimental anatomy at New York University Medical School, made this possible through their co-operation. The cadaver of a recently deceased infant was used. The internal iliac arteries were bilaterally ligated. The abdominal aorta, above the inferior mesenteric artery, was injected with barium solution. An x-ray was then taken to observe what collateral circulation had taken place. Perineal circulation is obviously present

From the Surgical Service, French Hospital.
Read in part and case presented at the New York Academy of Medicine December 3, 1937.
[1]Ann. Surg., 1927, September.

through collateral branches. Examination of the injected cadaver showed the uterine arteries definitely filled with barium, apparently through the ovarian arteries. This work is being carried further and will be reported later.

Dr. Shafiroff pointed out that collateral circulation, following ligation of the internal iliacs, is supplied in the following manner:

1. The uterine arteries which come from the internal iliac arteries, anastomose respectively with the right and the left ovarian arteries.

2. The superior hemorrhoidal arteries, arising from the inferior mesenteric artery, anastomose with the inferior and middle hemorrhoidal arteries, which come from the internal iliacs.

3. The pubic branches of the obturator artery, which is a branch of the internal iliac arteries, anastomose with the inferior epigastric arteries, which are branches of the external iliacs.

4. The circumflex and perforating branches of the deep femoral artery, have collateral branches with the inferior gluteal arteries, which come from the internal iliacs.

5. The superior gluteal arteries from the internal iliacs anastomose with the lateral sacral artery.

6. The ilio-lumbar arteries from the internal iliacs, anastomose with the lumbar arteries, which come from the aorta.

7. The lateral sacral arteries anastomose with the middle sacral artery.

8. The vesical arteries receive branches from the uterine and vaginal arteries.

The value of ligating the internal iliac arteries in operations for the removal of the rectum in carcinoma cases is well illustrated by a case under supervision in the spring of 1937.

An adult, white female, 64 years old, presented herself with the statement that she was suffering from hemorrhoids which were bleeding, and she wished relief from the bleeding, and assurance that there was no further trouble. Questioning elicited the fact that bleeding had been free for 2 months, and that she had lost 20 pounds in 3 months. There was the further history of unsatisfactory evacuation of the bowels and that no formed stools were passed. The patient did not complain of any pain shooting down the legs nor did she complain of backache.

Physical examination disclosed that the patient was pale, thin, nervous, and apprehensive. The patient weighed 158 pounds, which figure represented 24 pounds less than her average weight. The blood pressure was 130–80, the pulse was 82, and the heart was regular, of fair force, and showed no adventitious sounds. The mucous membranes were distinctly pale. A digital examination allowed the examining finger to enter about 3 inches, at which point there was a large, irregular fungoid growth filling the entire rectal lumen, with a passage through the center of this mass so small that it accommodated only one finger. The mass bled freely on digital examination. Proctoscopic examination confirmed the digital examination and disclosed the mass to be so friable that tissue was dislodged by the end of the proctoscope. The pre-operative work-up showed the blood count as follows: hemoglobin, 65 per cent; erythrocytes, 3,900,000; leucocytes, 9,000; polymorphonuclear neutrophiles, 61 per cent; lymphocytes, 37 per cent and monocytes 2 per cent. The Wassermann test was negative. The blood chemistry disclosed a blood sugar of 102 and non-protein nitrogen of 29. The x-ray confirmed the digital and proctoscopic examinations and showed the mass to be about 3 inches long, practically obstructing the entire lumen of the gut throughout the entire length of the growth. Roentgenograms of the lungs, the spine, and the other bones did not disclose metastases.

A preliminary left-sided colostomy was performed on May 17, 1937. At the time of this colostomy, the liver and other abdominal organs, were examined for metastases but no evidence was apparent. A period of 28 days was allowed to elapse while the intestines were decompressed, and the patient's general health was built up.

On June 14, 1937, the patient was transfused, and the growth was approached through a suprapubic midline incision under general anesthesia. The continuity of the descending colon and sigmoid were terminated just below the left colostomy and the proximal end was invaginated as was the distal end, which was then carefully protected to prevent further contamination. The mesocolon from this point, down to the promentory of the sacrum, was ligated and cut. At this stage in the operation, the step which was of particular interest was then carried out. The posterior peritoneal covering of the common iliac artery, on the right side, was incised at the bifurcation. The internal iliac was picked up from within out, by carefully passing a curved aneurism needle around it. The internal iliac was clamped, doubly ligated, but not cut. The same procedure was than carried out on the left side, thus stopping all pelvic circulation from the internal iliac arteries. A wide bilateral dissection on either side of the rectum and rectosigmoid was then carried out, down to the division between the rectum, and the uterus, and the vagina. The sigmoid was then grasped, and the mesocolon of the sigmoid was put under tension, as was the mesorectum. The mesocolon was then cut and practically no bleeding was encountered. A sponge, on a sponge stick, was used to free the loose cellular tissue in the curve of the sacrum. This gave complete exposure down to the growth, and it was then possible to pass the hand completely around the rectum, as there were no adhesions to the uterus, or to the vagina, or to any of the lateral structures, due to the bilateral dissection. The absence of bleeding was striking, and there was absolutely no problem involved in hemostasis. It was not necessary to ligate the middle sacral artery. Clamps were placed across the anal canal, well below the growth. The anal canal was cut across with a phenol knife, and the rectum together with the growth and the sigmoid were removed through the abdominal incision. The proximal end of the anal canal was whipped over and buried under a purse-string suture. Drains were placed in the pelvis and to the distal end of the descending colon, and the wound was closed in layers. The patient left the table in good condition. Postoperative course was not unusual and was helped by infusions and irrigations through the colostomy, which had been established for 28 days. The patient is alive and well and so far has justified the procedure after 8 months.

The points of particular interest are:

In certain cases it is possible to ligate both internal iliac arteries, without jeopardizing the life of the patient.

Thus, in selected cases, the rectum may be removed from above without danger of hemorrhage.

The patient is saved a prolonged illness, which would result if sacral or perineal approach were required.

Finally, the patient is spared the shock, the pain, and a prolonged healing process, which would be required in the posterior approach.

While the procedure of ligating the internal iliac arteries in surgery of the rectum and rectosigmoid is not the method usually practiced, our case proves that it is of invaluable assistance in selected cases. Leaving the perineum intact also violates the fundamental thesis of Miles that all three paths of lymphatic spread should be thoroughly extirpated even in early cases of carcinoma. However, violating Miles' thesis has been successfully done by such men as Mayo, Rankin, Jones, Lahey, and others, who have led the way in rectal surgery in this country in recent years. Leaving the perineum intact is further justified by the work of Westhues.[1] He disputes Miles' thesis of the downward spread of cancer of the rectosigmoid. In support of this he made most extensive microscopical examinations on the pathological tissues removed in 102 different cases of cancer of the rectum, done by the radical abdominoperineal method, which included removal of the sphincter. In only one case did they find metastases below the lower edge of the neoplasm. This will prove to be either a very helpful suggestion, or disastrous for some patients if incorrect.

Further search of the literature on ligation of the internal iliac arteries discloses the fact that this operation was performed by Dr. Howard C. Taylor, Sr., and his associates in 1924. This was done to control bleeding in a case of carcinoma of the uterus.

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# A MODIFIED WELL LEG TRACTION SPLINT AND DISTRACTOR COMBINED

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THE use of the well leg as a means of counter traction in certain fractures of the femur, and pelvic bones, has been established as a sound principle and a popular method of treating these cases since Anderson (1), published a description of his practical and ingenious splint. Other splints, somewhat similar in design, have appeared on the market since that time.

It is not the purpose of this article to relate further evidence of the effectiveness of this type of splint but to describe a modified well leg traction splint devised and used by the writer for the past 2 years, which in addition to possessing certain advantages over other such splints is also an efficient distractor.

## DESCRIPTION OF THE SPLINT

The writer's splint is in two parts, one known as the tractor, Figure 1, and the other as the retainer or immobilizer, Figure 2.

The tractor is composed of two upright metal bars, *1* and *1*, attached to removable plates, *2* and *2*, for incorporation in plaster-of-Paris. Each upright bar is attached to the cross arm, *3*, by a sleeve so that it can be moved along to any desired position, to fit any size leg, there to be anchored by thumb screws, *4*. At one end of the cross bar, *3*, there is a pivot, *5*, and on the other end a swivel, *6*. To the long cross bar, *3*, a lever, *7*, is attached through an eye which fits over the pivot, *5*. This lever is adjusted for right or left leg simply by detaching and turning it over. It is composed of a short arm and a long arm, being in relation of approximately 3 to 1. At the long end of the lever, *7*, there is a threaded bolt which passes through the swivel, *6*, and carries a stout spring, *9*, and a wing nut, *8*. In the short end of the lever are several holes through which an S-hook, *10*, a bolt or a piece of stout wire can be passed to connect the lever with a Kirschner bow, as will be explained.

The retainer, or immobilizer, consists of two metal bars *a* and *b*, both of the extension type. These bars, at their free ends, are connected by hinges to T-shaped metal bands, *1* and *2*, for incorporation in plaster-of-Paris. At the other end, *4*, these bars are connected by a hinge, but the shorter, or cross bar, extends slightly beyond the hinge joint to connect with a quadrant, *5*. The quadrant, *5*, in turn is connected to the third T-shaped metal band through a hinge joint. The quadrant permits rotation of the injured member, cross bar *b* permits the desired separation of the feet while diagonal bar *a* controls the extension as explained below.

## METHOD OF APPLICATION

*Fractures of the femur and pelvic bones.* A plaster-of-Paris cast is applied to the well leg, as for other types of well leg traction splints, extending from the toes to mid thigh. In addition to the pads over the knee, malleoli, and sole of the foot, a pad is placed over the inner side of the knee to prevent possible pressure from the metal T-band, *1*, of the retainer in case pressure is applied before the cast is hard. This cast is allowed to become reasonably firm and then the retainer is attached by securing the metal T-bands, *2* and *1*, just above the internal malleolus and opposite the inner condyle of the femur, respectively. These are held in place by several turns of plaster-of-Paris bandage. Some form of light anesthetic is now given and a Steinmann pin 8 inches long is drilled through the lower end of the tibia and a Kirschner wire through the lower end of the femur of the injured leg. Of course, a Steinmann pin may be used here instead of the Kirschner wire, if preferred. A well padded plaster-of-Paris boot is now applied extending from the base of the toes to the knee and surrounding the lower pin. As soon as the cast is firm the retainer is secured to it by attaching the third metal T-band, *3*, just above the internal malleolus with the Steinmann pin passing through the small hole located in the center of the cross arm of the metal band, *3*. When this is securely bound with plaster-of-Paris to the plaster boot, manual traction is gently applied to the injured leg with the wing nut, *7*, on the long diagonal bar *a*, unscrewed. As soon as the leg is pulled down as far as possible, wing nut *7* is tightened, thus holding what has been gained by traction, and wing nut *8* on the quadrant is unscrewed, the foot is rotated into the position which is desired and the wing nut *8* is then tightened again (Fig. 3).

The tractor is now applied to the well leg, plates *2* and *2* fitted on the cast of this leg between the attachments *1* and *2* of the retainer. A few turns of plaster-of-Paris or surgical gauze is sufficient to hold this in place. Some form of Kirschner bow is now attached to the wire in the femur. If a Steinmann pin is used instead of a Kirschner wire, the bow is unnecessary. Wing nut *8* of the tractor is now run out as far as necessary on its threaded bolt to allow connection between the Kirschner bow and S-hook, *10*. Traction is obtained by screwing down on wing nut *8* after unscrewing wing nut *7*, on the diagonal bar *a* of the retainer (Fig. 4).

In this splint an attempt has been made to overcome the objections commonly raised against well leg traction splints, namely: (1) the risk of permanently loose joints or habitual dislocation due to pull *through* the knee joint (3); (2) risk of overextension due to constant steady pull (1); and (3) pain in the hip joint of the well leg due to excessive pressure against these structures (1). In the writer's splint the first two of these objections have been overcome and the third has been reduced to a minimum, being absent entirely in most cases.

*Direct pull.* It will be evident from the above description that the pull is directly on the fractured bone, the femur, which we believe to be preferable to a pull through the knee joint with its attendant risk of stretching the joint structures. If the fracture is too low down in the femur, however, the pin, or wire is placed through the upper end of the tibia and the apparatus is attached with the knee of the injured leg slightly flexed (Fig. 5).

*Risk of overextension eliminated.* In this, as in any method of constant, steady traction, shortening is frequently overcome within a few days and satisfactory reduction is obtained. With the traction part of the splint separate from the immobilizing part, as in this splint, constant steady traction can be discontinued at any point and the tractor removed leaving only the retainer as shown in Figure 3. In this way we have eliminated the risk of overextension of the fragments with its danger of delayed, or perhaps non-union. As soon as sufficient traction has been obtained wing nut *7* on the retainer is tightened, wing nut *8* on the tractor loosened and the tractor, Kirschner bow, and wire (or Steinmann pin if used) through the femur are removed. The stress is now thrown on the foot of the injured leg but because of the Steinmann pin incorporated in the plaster and passing through the metal splint, the pressure which is put on the heel and dorsum of the foot is negligible.

As described by Anderson, traction by this method results in abduction of the injured leg and adduction of the well leg. With the two legs in this position and the fractured leg drawn out to its normal length, it will appear longer than the well leg. A cross bar connecting the two inner malleoli forms an *acute* angle with a diagonal bar extending from the inner malleolus of the fractured leg to the inner condyle of the femur of the well leg. As long as this angle remains acute, shortening of the fractured leg cannot occur unless the patient increases the abduction angle at the hip, which he is not likely to do. The usual inclination is to straighten up. This acute angle is obtained by the tractor and maintained by the cross bar *b* and the diagonal bar *a* of the retainer which become rigid bars when wing nuts *6* and *7* are tightened. This is the principle of the retainer.

*Pain in the hip of the well leg eliminated.* Considerable pressure can be exerted against a hip when the leg is in abduction without causing the patient discomfort because the pressure is against the bony framework, the acetabulum. With the leg in *adduction*, however, the pressure is more against the capsule and ligaments and causes considerable discomfort at times. This objectionable feature of well leg traction splints is mentioned by some writers and suggestions are offered for its remedy. Anderson (2) states that in some cases the pain is so severe it may "demand temporary release of traction." In the writer's splint this objectionable feature has been practically eliminated by placing the fulcrum much nearer the end of the lever to which the weight (traction) is attached than it is to the other end where the power is applied. The relative length is about as 1 is to 3, hence the pressure on the well hip is only one third as great as the pull on the injured leg. This amount of pressure is not enough to produce noticeable discomfort.

*Convenience.* The fact that this splint is applied within the "confines" of the patient has made it possible to use it where other types were not practical. One such instance was the case of a man 6 feet 7 inches tall with a compound fracture of the left femur. Even with the head end of the bed cranked up it was difficult to get this man into the regulation hospital bed. There was no room to add 6 inches of splint beyond his feet. The writer's splint was employed successfully in this case.

*Economy.* Undoubtedly the well leg traction splint often effects a saving to the patient in hospital expense because he can be moved to his home much earlier. However, the number of patients treated in any one hospital or by any one

Fig. 1. Tractor fully described in text.

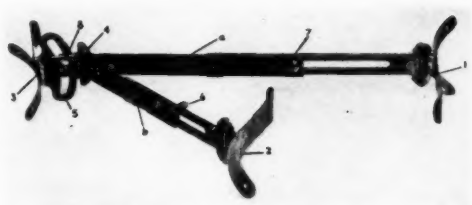

Fig. 2. Retainer or immobilizer.

doctor is limited to the number of splints the hospital or doctor can afford. In the writer's splint the patient is dismissed wearing only the retainer (Fig. 3) which is a very simple inexpensive device. Personally, we have had as many as five retainers on patients at the same time, most of them out of the hospital and the tractor available for use should another case come in. In this instance one tractor served five retainers.

Not infrequently cases are brought into the hospital, with fractures of the femur, who live quite a distance away and are not able to pay even a small hospital fee. Is the hospital to carry such a patient until the fracture is healed, or put a splint on him and send him on his way? With a retainer on such a case, especially if the plaster-of-Paris cast is extended up to the hips on each leg and braced across the top with a wooden strip incorporated in the plaster, such a case can be transported great distances safely. It would be a great saving to the hospital though the retainer should never be returned. One would hesitate, however, to send such a patient away with an expensive splint on without a cash deposit, and the type case referred to is unable to make that deposit.

Fig. 3. Retainer applied; ready for tractor. This also illustrates the appearance of the splint when patient is dismissed.

*Only one size necessary.* Due to the fact that this splint can be adjusted to fit both adults and children, it is necessary to have but one size. The economy of this feature is at once apparent.

I have already indicated that well leg traction splints in general are often economical splints and that this one in particular is especially so. In support of this statement, let me mention other uses of this splint which make it almost a universal one as far as the long bones are concerned. This feature makes it unnecessary to have an expensive outlay of arm and leg splints.

### FRACTURES OF THE LOWER LEG

Figure 6 illustrates how the tractor is used as a distractor for reducing fractures of the lower leg, by attaching a simple extension bar which pushes on the Kirschner bow. A Steinmann pin is drilled through the upper and lower fragments of the

Fig. 4. Tractor and retainer applied in a case of compound fracture of the right femur. Diagonal bar of retainer disconnected.

Fig. 5. Fracture lower end of left femur. Splints applied showing how flexion is obtained for this type of fracture. This picture was made just before tractor was removed, hence the plaster-of-Paris extension above the knee of left leg.

tibia. These are incorporated in plaster-of-Paris, leaving a gap at the fracture site; or the entire leg may be incased in plaster-of-Paris and a circular cut made half way between the two pins as suggested by Miller. The tractor is then fitted on the upper cast with this pin passing through holes in members *2* and *2*. A few turns of plaster will hold this in place. A simple extension bar, possessing an attachment for rotation in case the two pins are not parallel, is now affixed to the long arm of the lever on the tractor by one end and to some form of Kirschner bow at the other. Screwing down on wing nut *8* produces effective distraction and the rigid bar makes a substantial support for the leg so that it can be moved about without pain to the patient and without fear of disturbing the fragments. If the wound is compound, dressings are easily done. As soon as reduction is satisfactory, small blocks of wood are placed in the gap, a posterior molded plaster splint is applied and allowed to set, after which the tractor is removed and a few turns of plaster makes a firm cast. The Steinmann pins are preferable to the Kirschner wires in these cases because of their rigidity.

### FRACTURES OF THE ARM

Figures 7 and 8 illustrate the use of the tractor as a distractor in cases of fractures of the humerus and both bones of the forearm. As shown in Figure 7, a shoulder spica of plaster-of-Paris is first put on with the arm held in the most favorable position as determined from radiographic films. The cast is carried far enough down the arm to afford room to attach the members of the tractor. Detachable members *2* and *2* are left off. A few turns of gauze bandage will hold the tractor securely. A 6-inch Steinmann pin is now

Fig. 6. Tractor converted into a distractor for reduction of fracture of both bones of the lower leg by attaching an extension rod to the long arm of the lever.

drilled through the ulna near the olecranon process (or through the lower humerus) and with the forearm at right angle to the upper arm a plaster-of-Paris cast is applied from the hand to, and including, the elbow, incorporating the pin. The extension rod is attached to the long arm of the lever as for the lower leg, and a small right angle bar of special design is fitted to this rod. Some form of Kirschner bow is attached to the pin and connected to the right angle bar as shown in Figure 7. The hand is supported by a sling from the short arm of the lever. The extension rod splints the fracture and rotation is provided for in the same rod. Extension of the humerus is accomplished in the usual way by screwing down on wing nut *8*. Reduction may be carried out at once, under fluoroscopic guidance, the gap between the casts filled in with plaster, after which the entire mechanism is removed. Reduction by this means is a great saving in hospital expense to the patient because of early dismissal.

Figure 8 illustrates one way of attaching the tractor for obtaining extension in fractures of the radius and ulna. The forearm is held at right angle to the upper arm, a plaster-of-Paris cast is applied to the upper arm, around the elbow and far enough down the forearm to form a base for the attachment of the two upright bars *1* and *1* of the tractor. The same push rod used for fractures of the humerus is attached by one end to

Fig. 7. Tractor used as distractor in reducing a fracture of the humerus.

the long arm of the lever, *7*, and by the other end to finger traps attached to the fingers. If skeletal traction is preferred a Kirschner wire or Steinmann pin is put through the lower end of the radius and to this is attached a Kirschner bow for connection with the right angle bar. Extension in either method is obtained by screwing down on wing nut *8*. Reduction can be carried out under the fluoroscope if desired and when satisfactory the cast can be continued over the rest of the forearm and hand and, when this is hard, the apparatus is removed. The extension bar necessary to convert the tractor into a distractor is simple and inexpensive when compared to the cost of the apparatus usually employed as a distractor. Most hospitals and surgeons doing fracture work are equipped with some form of Kirschner bow, hence no added expense is incurred here.

### FRACTURES OF THE NECK OF THE FEMUR

*Intertrochanteric.* Experience has shown that most of these fractures result in bony union if securely held for 10 to 12 weeks, sometimes earlier. Hence we employ the well leg splint in these cases. The "retainer" is applied in the regular way and reduction made by manual traction at once and the wing nuts tightened; or the routine for fractures of the shaft may be employed.

*Intracapsular fractures.* Most writers, including Magnuson, express disappointment over the results obtained in these cases treated with a well leg splint. Junkin, however, reports 38 cases of hip fractures treated in this way, 15 of which were

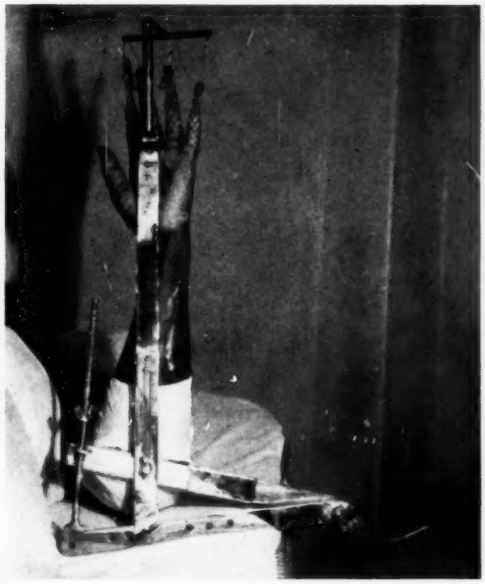

Fig. 8. Manner of applying the tractor for reducing fractures of the forearm; or a pin may be put through at the wrist and a Kirschner bow applied.

intracapsular and "all living past a year recovered with bony union and good function." Magnuson suggests that failure might be due in part to continuous traction. By using only the retaining part of the writer's splint in those cases following reduction by the Ledbetter method, it is an easy matter to maintain good position of the fragments, but failure of union often results despite the fact that there is *no traction at all* with this splint used in this way. Some of these cases may develop bony union as late as a year but it is not practical to keep a splint on that long. Hence this splint is not used for intracapsular fractures unless they are of the impacted, incomplete type. This type, it is true, will usually heal firmly if the patient is left alone in bed without splints of any kind but there is the danger of completing the fracture. Besides there is usually a certain amount of external rotation of the foot. Application of the retainer in the usual manner, *without* the insertion of a pin or wire (since there is no muscle pull) and with internal rotation of the foot to 20 degrees, enables the patients to get out of bed and into a chair without danger of completing the fracture and when healing is completed the external rotation of the foot has usually disappeared.

SUMMARY

A combination well leg traction splint and distractor is described, the technique for its application is given in detail and its several advantages set forth.

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# ABERRANT PANCREATIC TISSUE IN THE GASTRO-INTESTINAL TRACT

## Report of Two Cases and Review of Literature

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ABERRANT pancreatic tissue is still popularly considered an infrequent anomaly. To Klob is given the credit of having described the first case in 1859 (2). Since that time a number of cases have been reported in the medical literature, particularly within the last decade. In most instances the finding was made at autopsy examination, apparently the tissue not having produced clinical symptoms denoting its presence.

The presence of aberrant pancreatic tissue is rarely suspected before operation; indeed, a careful perusal of the reported cases shows that, with few exceptions, its presence was not recognized at the time of operation. The pre-operative symptoms resulting from aberrant pancreatic tissue were usually interpreted clinically as being caused by peptic ulcer, by cholecystitis, or even by malignancy.

The first cases recorded in the literature in which the syndrome of the symptoms produced by aberrant pancreatic tissue was given presumably as the indication for operation, were those of Hulsi, Leyden and Reynier and Masson 1909 (6). Hulsi found pancreatic tissue in a Meckel's diverticulum. In the cases of the others operation was undertaken to relieve suspected cancers of the pylorus; during the course of the operation, however, aberrant pancreatic tissue was found which without doubt had been the cause of the clinical symptoms. Many similar observations have since been made, much to the chagrin of the surgeon who, in the course of operation, for instance, in the case of a suspected duodenal ulcer or some other upper abdominal condition, discovers aberrant pancreas or aberrant pancreatic tissue which would explain the patient's symptoms.

From the Surgical Service, Beth Israel Hospital.

In his review of 43 cases in which aberrant pancreas were found at the time of operation, Pappi states that the most frequent pre-operative diagnosis was either gastric or duodenal ulcer. This diagnosis was made in 17 cases, or 39.5 per cent. The aberrant pancreatic tissue in these 17 cases was found in the stomach, jejunum, gall bladder, and Meckel's diverticulum. In 10 of the 43 cases, no specific diagnoses were made; 7 were either diagnosed as stones, hepatic colic or were vaguely described as "pathology" in the right hypochondriac region. The most common site of the aberrant pancreas in these 10 cases was either on the duodenal or gastric wall.

In this connection it is interesting to note that aberrant pancreas was considered, by some, as a possible etiological factor in the causation of duodenal ulcer. Scagliosi, quoted by Pappi, states that, from several cases examined by him, he was led to the conclusion that the aberrant nodules inclosed in the intestinal wall may produce an ischemia in this area through its compression effect which causes an atrophy of the mucosa and by its mechanical action may bring about atrophic disturbance of the nerve filaments, thus leading to the formation of ulcer. Some believe that the digestive property of the pancreatic tissue may

cause an ulceration of the intestinal or gastric mucosa; however, these are purely academic discussions and no definite proof has been brought forth to substantiate this theory.

It is the commonly accepted belief that aberrant pancreas is of congenital origin. The objection to this theory is mainly based upon the following reasons: (a) the finding of pancreatic tissue during postmortem examinations of newborn children is very rare, and (b) most of the patients, in whom this condition was found at the time of operation, were of the second decade of life or older; however, these objections to the theory of congenital origin do not seem to be valid in the light of recent postmortem observations.

In a series of 19 postmortem examinations reported by Branch and Gross, 5 instances, or 25 per cent, were in newborn children, the ages varying from 8 weeks to 3 months, a fact showing definitely that the presence of this tissue in the newborn is not as infrequent as it is thought to be; furthermore, one must take into consideration the fact that while aberrant pancreatic tissue is easily recognizable macroscopically in postmortem examination of an adult, because in the adult it has attained a fair size, it can be diagnosed only on microscopic examination in the newborn. Therefore, in infants, it frequently goes unrecognized in the ordinary postmortem inspection. Such tissue may remain dormant for many years in the early life of an individual and then suddenly give rise to clinical symptoms of varying intensity.

The relative frequency of the presence of aberrant pancreatic tissue based upon the finding in 4,076 autopsies reported by 6 different pathologists (6), varies between 0.55 and 3 per cent, the average being 1.89 per cent. The most common site is the stomach and duodenum, about equally divided—31 per cent each, the next most frequent locations are the jejunum, 21 per cent; ileum, 9 per cent; peritoneal lining, 3.37 per cent; and the least common location of this condition is the gall bladder and spleen, 1.48 per cent and 0.74 per cent, each.

Aberrant pancreatic may be generally divided into two types: (a) the annular pancreas and (b) the aberrant pancreatic nodule.

*The annular pancreas* is a rare developmental anomaly in which the viscus is situated normally but encircles in a ring-like fashion the second portion of the duodenum, thus giving rise to a constriction of that portion and a dilatation of the portion above it. This condition has a distinct clinical significance, because of its tendency to cause stenosis, and in some instances it has produced all the signs and symptoms of high intestinal obstruction. McNaught and Cox collected from the literature and tabulated 44 cases; in 9 of these cases signs and symptoms of high intestinal obstruction of various degrees were present. Therefore, this condition must be kept in mind whenever symptoms of high intestinal obstruction present themselves. In most of the cases in this group the annular pancreas were incidental autopsy findings. There was nothing in the history of the patients to indicate that this anomaly caused them to suffer any gastro-intestinal discomfort during life. The symptoms of acute intestinal obstruction which result from such an anomaly probably arise after "swelling of the pancreatic ring through hemorrhage, pancreatitis, tumor, hypertrophy, etc.," have taken place.

*Aberrant pancreatic nodules.* A more common form of aberrant pancreatic tissue, the one with which this communication is chiefly concerned, is the small nodule varying in size from several millimeters to 6 centimeters in diameter. Such nodules may be situated either on the wall of the stomach, duodenum, jejunum, or in an intestinal diverticulum. Several observers have reported the presence of small nodules in the hepatic duct, the gall bladder, and omentum. The most common site, however, is the duodenum, the jejunum, and the walls of the stomach, as stated above.

The various theories as to the origin of the accessory pancreatic tissue have been thoroughly covered by other writers and will not be repeated here. Our purpose in writing this paper is to report 2 more cases of this anomaly and to present a brief review of the literature. The importance of bearing in mind this condition as a distinct clinical entity in the interpretation of symptoms referable to the gastro-intestinal tract and of recognizing it at the time of operation will be clearly shown from the findings in these cases.

Many surgeons, including the writer, have had the unfortunate experience of mistaking aberrant pancreatic tissue for a carcinomatous lesion of the stomach or intestine. Considerable local inflammatory reaction at the site of aberrant pancreatic nodules will produce symptoms simulating those of pyloric or duodenal ulcer and at times even at operation the true condition will not be recognized.

In several recorded instances, risky operative procedures could have been omitted if correct diagnoses had been established either before operation or at least at the time of operation. In 10 of the 28 cases reported in the most recent American literature aberrant pancreatic tissue was

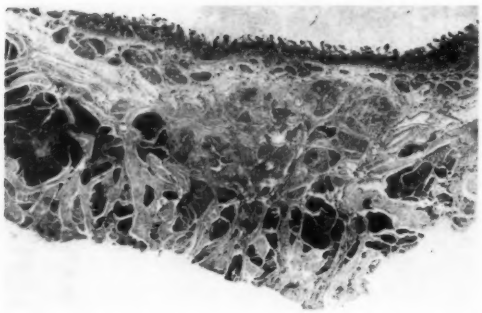

Fig. 1. Case 1. Pancreatic tissue and ducts in wall of duodenum. Low power.

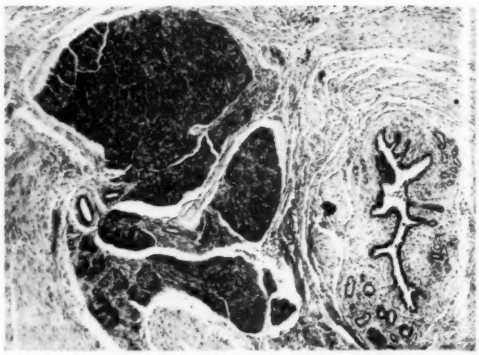

Fig. 2. Case 1. Aberrant pancreatic tissue and ducts containing leucocytes. ×125.

an operative finding; in the remaining 18 cases, the diagnoses were made at autopsy (1, 2, 3). The diagnosis was not made before operation in any of the 10 cases, nor was the true condition recognized even at the operating table, except in 1 case (8); its presence was only suspected at the time of operation in another case. In 2 of these cases, partial gastric resection for a supposed carcinoma of the pylorus was done. Hemi-pylorectomy was performed in 2 other cases in which a pre-operative diagnosis of duodenal ulcer was made and the exact condition was established only by microscopical examination of the tissue removed at operation. A pre-operative diagnosis of gall-bladder disease was made in one of these cases and cholecystectomy was done. A small tumor was observed in the stomach wall which was excised, and upon microscopical examination proved to be pancreatic tissue. A resection of the pylorus was done in another case for pyloric obstruction of a supposedly inflammatory nature. In one case a pre-operative diagnosis of appendicitis was made, but at operation a Meckel's diverticulum containing aberrant pancreatic tissue was found. In another case in which an operation for proved carcinoma of the stomach was done, aberrant pancreatic tissue was incidentally found in the duodenum. The true condition was recognized at the time of operation in one case in which a tumor, the size of a hazel nut, was removed from the anterior duodenal wall, with favorable post-operative recovery.

It is therefore important to sound another note of warning to the unwary of the existence of this condition not so commonly known. Many surgical pitfalls are caused by anatomical anomalies which the surgeon encounters in the course of an operation; constant reiteration of the existence of such anomalies will, in many instances, prevent unfavorable postoperative results. This can be done most impressively and forcefully by reporting the surgical instances in which such anomalies have been encountered but not recognized. In many such cases the end-results would have been obviated if the true condition had been recognized at the time of operation.

Up to the present writing, 328 cases of aberrant pancreatic tissue were recorded in the literature; this does not include the 44 cases of annular pancreas reported by James B. McNaught—a condition which is more easily recognized at the operating table than the irregularly distributed nodules of aberrant pancreatic tissue along the gastro-intestinal tract. A most comprehensive report of this condition, with a careful tabulation of 300 cases reported in the literature was published by Arrigo Pappi (2). Since the publication of his article, reports of 28 other cases have appeared in the literature (1, 2, 3, 8).

## CASE REPORTS

Case 1. B. K., white male, age 45 years, was admitted to the hospital on November 20, 1935. His chief complaint was rectal bleeding. Family and past history were irrelevant. Present illness began about 3 months before admission, when he noted several drops of blood in the stools. Since that time he had noticed, on several occasions, large amounts of blood in the stools which, at times, were dark red and sometimes black. He never had any sharp upper abdominal pain, nausea or vomiting. He had short periods of diarrhea, having four or five stools per day, which were followed by periods of constipation. For 3 weeks prior to admission, the patient had no rectal bleeding, but did have a dull ache over the entire abdomen. The patient gave no history of anorexia or loss of weight and his general physical examination, at the time of admission to the hospital, was essentially negative.

Report of gastro-intestinal series, completed on November 28, showed "probably duodenal ulcer with some pyloric retention." Barium enema showed some narrowing

at the rectal ampulla. Stool specimens on December 6 and 7 were strongly positive for occult blood. The patient was put on a Sippy diet after admission and felt fairly comfortable for a short time. A diagnosis of duodenal ulcer with possibility of early malignancy was made and operation was advised.

The operation was performed on December 11. Through an upper right rectus incision the abdomen was explored; the gall bladder was found to be normal; the stomach was normal; the pylorus appeared hard and retracted near the duodenal junction. On the first portion of the duodenum was found a small nodular growth of a cauliflower appearance, firmly incorporated in the duodenal wall, close to the retracted area. This finding, in conjunction with the history of occult blood and x-ray findings, led us to believe that we were probably dealing with a peptic ulcer and the possibility of early malignancy was also considered. A radical operation was therefore decided upon and a partial gastric resection of the Pólya type was done.

The postoperative course ran along very smoothly for the first 6 days. There was no vomiting or distention; the patient retained all fluids. On December 17, 6 days following operation, the patient vomited a foul-smelling reddish fluid; this was followed by some abdominal distention which was relieved by Levine tube. On the ninth day after operation, the temperature, which had never been above 101 degrees suddenly rose to 103.8 degrees and the patient went into sudden collapse. The pulse became rapid and thready and the skin cold and clammy. He remained in this condition until the next day when he went into extreme collapse and died on December 22, the eleventh day after operation.

Surgical pathology report by Dr. William Antopol: The gross specimen consists of resected portion of stomach and proximal 1 centimeter of duodenum. The entire specimen measures 11 centimeters along the greater curvature and 4 centimeters along the lesser curvature. The mucosa of the duodenum is markedly injected and granular. There are clamp marks on the duodenal portion which obliterate a good deal of its architecture. Along the line of extension of the lesser curvature and incorporated in the wall of the duodenum extending to the submucosa, there is a nodule which is 1 centimeter in its circular diameter and 8 millimeters thick. On cut section this has the appearance of glandular elements in a fatty stroma.

*Microscopical examination.* Mucous membrane, in several places, is absent. In the mucosa, there are congested areas as well as areas containing an increase in polymorphonuclear leucocytes. Numerous islands of pancreatic tissue and ducts are present through all layers, including the mucosa, and extending outward so as to form a projection of the serosa. In these areas, the muscular bundles are sparse, the Brunner glands are displaced. There are also zones in which the ducts are almost the sole epithelial constituents. Some of these ducts are dilated and contain numerous polymorphonuclear leucocytes. An occasional duct shows a squamous cell metaplasia of the lining cells. The corresponding interstitial tissue contains many fibroblasts and polymorphonuclear leucocytes. A rare island of Langerhans is seen.

Microscopical diagnosis: Aberrant pancreatic tissue in the wall of the proximal portion of the duodenum with chronic duodenitis (Figs. 1 and 2).

*Autopsy findings.* Abdomen: Peritoneal cavity presents a grayish and greenish yellow stringy and purulent exudate at the root of the mesentery. The serosa of the proximal loops of the jejunum is thickened and dulled in appearance and it is covered by small and large flakes of stringy yellow material.

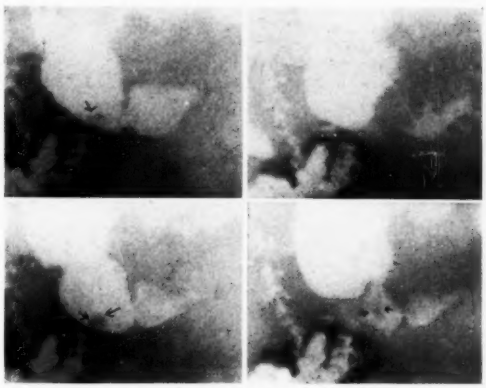

Fig. 3. Case 2. X-ray showing benign papilloma of pars pylorica.

Gastro-intestinal tract: The antral portion of the stomach and the proximal few inches of the duodenum have been resected. The distal end of the stomach is found tightly sutured to a loop of jejunum. The gastro-enterostomy appears to be well closed and no opening can be seen in the line of suture. The proximal end of remaining duodenum is covered with a shaggy, dull, greenish black material which encroaches on the wall of the duodenum and onto the origin of the root of the mesentery, in the region of the ligament of Treitz. The duodenal stump appears well closed except at one point where there is a small opening about 3 millimeters in diameter which is partially plugged with greenish black material. The ascending colon is covered with patchy exudate of stringy, yellowish-green material.

Diagnosis: Leakage from duodenal stump with fibroid purulent peritonitis. Also localized patchy pneumonia right middle lobe and right lower lobe.

Case 2. S. S., white female, aged 45 years, was admitted to the hospital on December 6, 1936, complaining of a continuous feeling of weakness and fatigue. This had been present for a period of several months. There had been no other complaints until 10 days ago, when she awoke with a severe headache which was accompanied by a feeling of nausea and repeated vomiting. No abdominal pain or distress was noted; a narcotic was administered, after which the acute symptoms disappeared. However, the sense of weakness and inability to do even light physical work seemed to be aggravated since the attack. Bowels, which were always regular, became constipated for several days following the acute incident; during this period for the first time she took cathartics. The remainder of her history was of no special significance.

Physical examination revealed an extremely obese female. A rubbing systolic murmur was heard at the apex which was transmitted toward the base as well as toward the axilla. The rhythm was regular; no other positive findings were noted. Hemoglobin on admission was 95 per cent; red cell count, 4,850,000; white blood cell count, 5,900. Urine was essentially negative; Wassermann blood and Kline tests were negative.

X-ray examination of the gastro-intestinal tract before admission to the hospital was reported as indicating "probably early gastric malignancy." Examination at the hospital by Dr. N. J. Furst showed "the stomach to be of

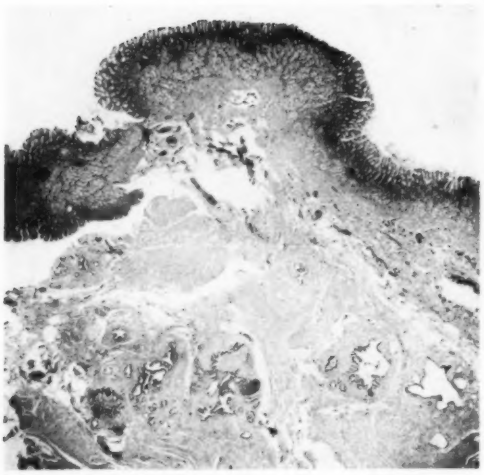

Fig. 4. Case 2. Polypoid lesion of stomach with pancreatic tissue and ducts within stomach wall. Low power.

medium large size, of active tone, of good mobility and separability. No defect in pars cardia or media was observed. On the greater curvature side of the pars pylorica, there was a more or less persistent area of circumscribed illumination, probably due to polyp formation (Fig. 3). Peristalsis was comparatively normal; gastric evacuation was delayed, resulting in retention of 40 per cent of the breakfast meal. At 24 hours, the major portion of the barium had been expelled precluding a morphological description of the colon. Conclusion: pyloric polyp, probably benign."

In view of the history of 10 days' persistent vomiting, followed by weakness and the report of the examinations of the gastro-intestinal tract, both before and after admission to the hospital, operation was decided upon. The pre-operative diagnosis was polyp of pylorus, probably causing pylorospasm and obstruction.

The operation was performed on December 11; the abdomen was opened by an upper right transrectus incision. The external appearance of the stomach and duodenum was normal. There were no adhesions between the gall bladder, pylorus and duodenum. The gall bladder was small, not distended; no stones. After thorough palpation of the stomach, a small, soft mass was felt within the lumen of the pylorus. The entire stomach was pulled down and held to the parietal peritoneum below the umbilicus by omentum which was fixed in that position, probably as a result of a previous operation. The pylorus was incised and the mass, with a segment of stomach wall, was removed. The incision was closed at right angles by two layers of catgut for peritoneum, interrupted chronic gut for fascia and silkworm gut for skin.

Postoperative course: Patient vomited some blood after operation and hemoglobin came down to 50 per cent. A blood transfusion of 500 cubic centimeters was administered, supplemented by intravenous injections of 5 per cent glucose during the next 48 hours. The subsequent postoperative course was entirely uneventful. The patient was discharged on January 4, 1937, in very good condition. The follow-up several months later showed patient to be free from any gastro-intestinal symptoms.

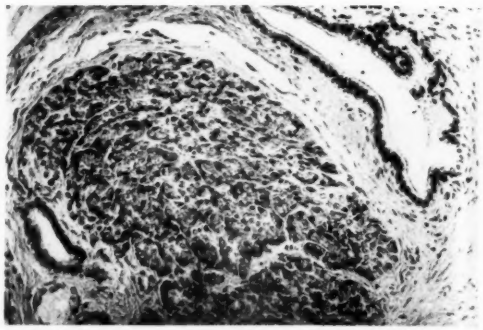

Fig. 5. Case 2. Aberrant pancreatic tissue and ducts. ×165.

Gross examination of the surgical specimen by Dr. William Antopol: The specimen is of polypoid structure, 1 centimeter in height and 1½ centimeters in diameter. On cross section it was seen to be composed of a grayish-yellow tissue arising from a broad pedicle. Numerous dilated "duct-like" lumina traversed the polyp and an occasional cellular area was seen in the muscularis and subserosa.

Microscopical examination revealed areas of pancreatic tissue through the muscularis and subserosa with dilated ducts in both the muscularis and submucosa. At times, these dilations reached cystic proportions. No islands of Langerhans were noted.

The diagnosis of polyp of the stomach with aberrant pancreatic tissue and distended ducts was made (Figs. 4 and 5).

The object of reporting these 2 cases is to bring again to the attention of the profession the fact that aberrant pancreatic tissue is not as infrequent as it is commonly believed to be.

The unfortunate postoperative outcome in Case 1 illustrates very strongly the importance of being able to recognize and evaluate the significance of this condition before operation, or at least at the time of operation. The absence of any other pathological condition in the gastro-intestinal tract in Case 1, as demonstrated by our operative exploration and subsequently supplemented by the post-mortem examination and the microscopical diagnosis of "aberrant pancreatic tissue in the wall of the proximal portion of the duodenum with chronic duodenitis" proved conclusively, to my mind, that the clinical symptoms were entirely due to this condition. We were strongly influenced in the choice of operation because of the presence of the pancreatic nodule which probably caused the local inflammatory condition of the duodenal wall in that area. The general appearance of this pancreatic nodule together with the indurated area around it led us to suspect early malignancy. A resection of the pylorus and the first portion of

duodenum was done. In view of the postoperative findings, it is extremely doubtful whether such a radical operation was a justifiable procedure in this case. A much simpler operation, such as gastro-enterostomy, could have probably relieved the patient's symptoms.

In Case 2, the aberrant pancreatic tissue with the enormously dilated glands undoubtedly was the cause for polyp formation. This was located in the prepyloric region and would act like a ball-valve. This might have been the cause of the pylorospasm, retention, and the onset of acute vomiting, which made the patient seek medical relief. The x-ray diagnosis of early malignancy and the subsequent more correct diagnosis of pyloric polyp were the deciding factors for surgical treatment.

The recognition of the benign nature of this structure, before and during the operation, obviated the necessity for any radical surgical procedure. A partial hemipylorectomy was performed and this procedure was followed by an uneventful recovery.

## SUMMARY

The report of 2 cases, in addition to those previously reported by other writers, in which the presence of aberrant pancreatic tissue along the gastro-intestinal tract was found, is presented. A brief review of the literature, indicating the infrequency with which this condition is diagnosed, either before or at operation, is given. The difficulty of making a correct pre-operative diagnosis and the importance of recognition of the condition, particularly at the time of operation, is pointed out so that such unfortunate results as were obtained in Case 1 may be avoided.

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# A RELIABLE CONTROL FOR STEAM STERILIZATION

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ALTHOUGH surgeons, well schooled in aseptic technique, recognize the basic importance of the sterilization of surgical supplies, they are not familiar with the actual technique of sterilization and assume it to be automatic and infallible. The responsibility for the preparation of surgical supplies has been delegated to others without the realization that those entrusted with supervision of the work have no reliable means of detecting faulty sterilization. More intimate knowledge of sterilizers and a brief consideration of the principles underlying the use of steam as a sterilizing agent emphasize the fact that safeguards must be utilized to insure the absolute sterility of surgical supplies.

The appearance of modern sterilizers inculcates trust in their performance, and satisfactory performance is conceded even though there is no objective assurance that the sterilizers have been operated properly. The enforcement of a theoretically safe sterilizing schedule may appear to eliminate faulty sterilization, yet neglect of proper inspection, cleansing or maintenance of the sterilizers may have impaired their efficiency so that sterilizing conditions are not actually attained. The hospital staff thinks of sterilizers as plant equipment, while the maintenance department considers them surgical apparatus, with the result that sterilizers are widely recognized (10) as being the most neglected of all hospital equipment.

The rapidity of its action and its wide applicability have established steam as the chief agent for the absolute sterilization of most surgical supplies. The fact that steam must be subjected to pressure to obtain temperatures destructive to bacterial life has made steam sterilization seem mysterious and its control difficult. The sterilizing efficacy of steam depends upon two factors—the *temperature of the saturated water vapor* in contact with the least accessible spore in the depths of the unsterile material, and the *duration of the exposure* to the hot vapor.

Steam penetrates and heats porous materials in the sterilizer because 81 per cent of its energy is in the form of latent heat. When steam is chilled it condenses and the latent energy of condensation is dissipated by heating the environs. Thus when steam strikes the outer layer of a relatively cold bundle of fabric, condensation occurs with consequent heating of the bundle. This exchange of heat continues, layer by layer, until the entire bundle is heated to the temperature of the surrounding steam and contains moisture equivalent to the quantity of heat liberated by the condensed steam. The penetration of the bundle is accomplished principally by the liberation of heat from steam carried into its depths by convection, not by the conduction of heat into its center.

Autoclaves and dressing sterilizers are merely steam pressure chambers arranged to provide ready access to their interiors. Each time the door of such a chamber is shut, air is trapped in a closed space. When steam under pressure is admitted to the space, the air is displaced since it does not mix immediately with the lighter, hotter steam which tends to stratify. When the gases ultimately become homogeneous through diffusion, each exerts its own partial pressure and the temperature of the mixture is lower than that characteristic of steam under the pressure indicated by the gauge (Fig. 1) and its heating efficiency is markedly decreased (Fig. 2). If pressure[1] were destructive to bacterial life, suitable conditions for sterilization would exist. However, pressure is but a means of raising the temperature of the steam sufficiently to coagulate bacterial proteins readily. The trapped air hinders sterilization because it prevents the development of the temperature characteristic of saturated steam under 15 pounds gauge pressure.

Before steam is admitted to a closed autoclave, the air entrapped in the chamber is at atmospheric pressure and is heavier than the steam which is released into the chamber. Hence the air gravitates to the bottom as the lighter, hotter steam enters. If the air can escape, steam quickly displaces most of the air; the pressure built up in the chamber is that exerted by the steam alone, and conditions of temperature and moisture are suitable for the rapid destruction of all bacterial life.

Modern autoclaves and dressing sterilizers are equipped with automatic valves which permit the air to escape from the bottom of the chamber as the steam displaces it. Fouling of the air ejector

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[1] A pressure of 3,000 atmospheres is not sufficient to kill Eberthella typhi, Escherichia coli, Mycobacterium tuberculosis, or Bacillus subtilis. Six thousand atmospheres pressure killed vegetative bacteria (3).

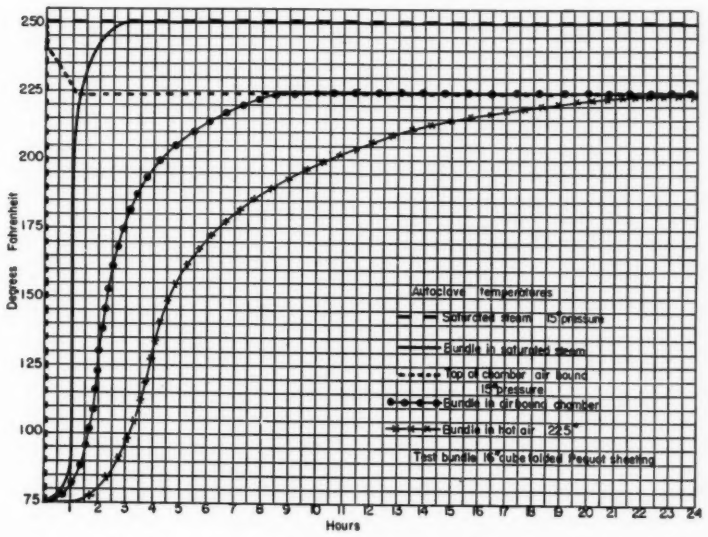
Degrees Fahrenheit
Hours
Autoclave temperatures
Saturated steam 15 pressure
Bundle in saturated steam
Top of chamber air bound 15 pressure
Bundle in air bound chamber
Bundle in hot air 225°
Test bundle 16 cube folded Pequot sheeting

Fig. 1. Temperature curves illustrating the time required for penetration of a 16 inch cube of Pequot sheeting under various conditions. The efficiency of saturated steam as a heating agent contrasts markedly with that of a mixture of air and steam (air bound chamber) or hot air alone. The curves were obtained on a recording potentiometer (Leeds and Northrup micromax).

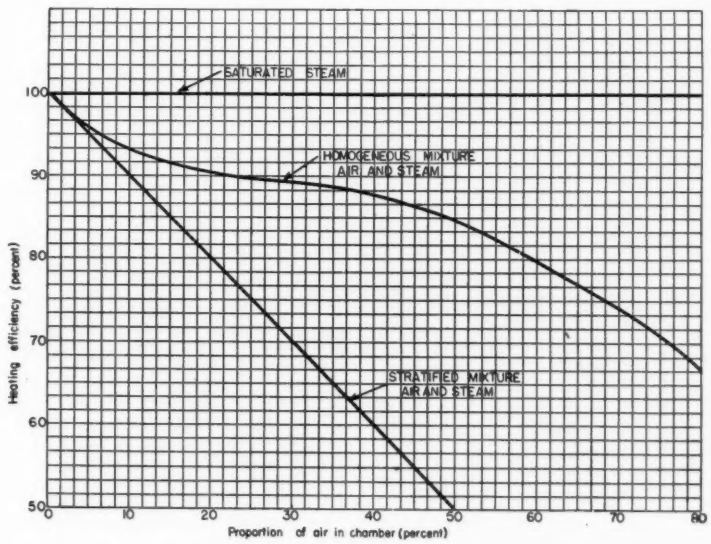
SATURATED STEAM
HOMOGENEOUS MIXTURE AIR AND STEAM
STRATIFIED MIXTURE AIR AND STEAM
Heating efficiency (percent)
Proportion of air in chamber (percent)

Fig. 2. The effect of air in the steam chamber on heating efficiency. (Chart courtesy Warren Webster & Company).

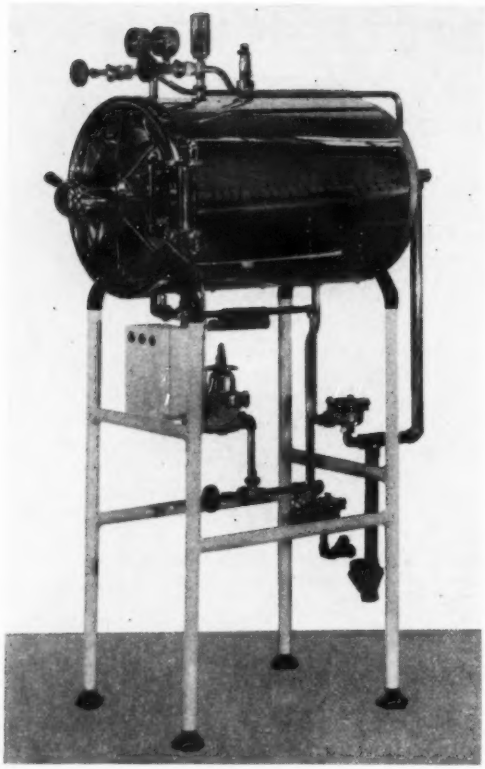

Fig. 3.

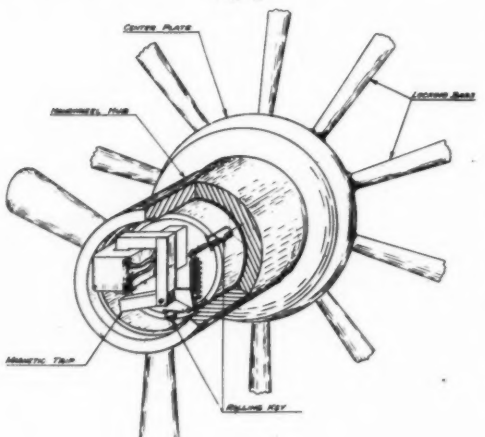


Fig. 4.

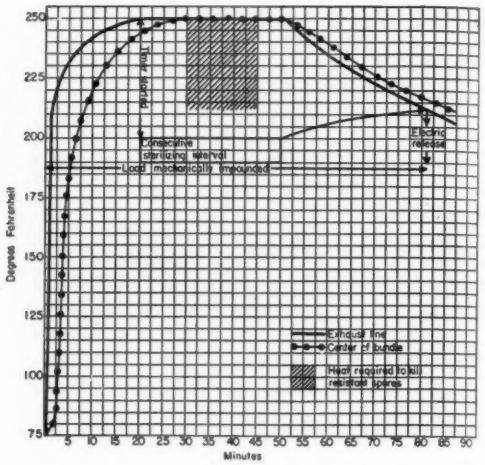


Fig. 5.

Fig. 3. A dressing sterilizer equipped with the control impounds the unsterile load until it has been exposed to saturated steam for a predetermined consecutive interval. Note the control box with its signals, the thermoswitch housing in the exhaust line, and the locking mechanism in the hub of the hand wheel.

Fig. 4. Cutaway drawing illustrating the rolling key clutch mechanism which wedges the handwheel so that the sterilizer door cannot be opened until the keys are released by a magnetic trip at the end of a satisfactory sterilizing cycle.

Fig. 5. Time temperature relationships which must be met during a sterilizing cycle before the load is released.

or exhaust line with lint, bits of rubber band, or paper tags, pins and other débris is a frequent cause of mechanical failure of sterilizers.

Just as air trapped in the chamber prevents the development of satisfactory conditions for sterilization, air confined within a bundle of surgical supplies hinders the convection of steam into the bundle. If the bundle is loosely packed the air can easily escape as it is displaced by the steam, and penetration with consequent heating of the bundle is rapid and complete. Tightly packed drums and overloaded sterilizers, however, require prolonged exposure to insure slow penetration by conduction.

The problem of controlling steam sterilization involves the determination of when *penetration is complete* and the enforcement of *exposure* of the load to an interval of heat destructive to bacterial life.

Numerous devices (2, 3, 6, 7, 8, 9, 13, 14, 15, 17) have been designed to indicate the temperature attained within a steam sterilizer or its load. Many sterility detectors have intrinsic faults (5,

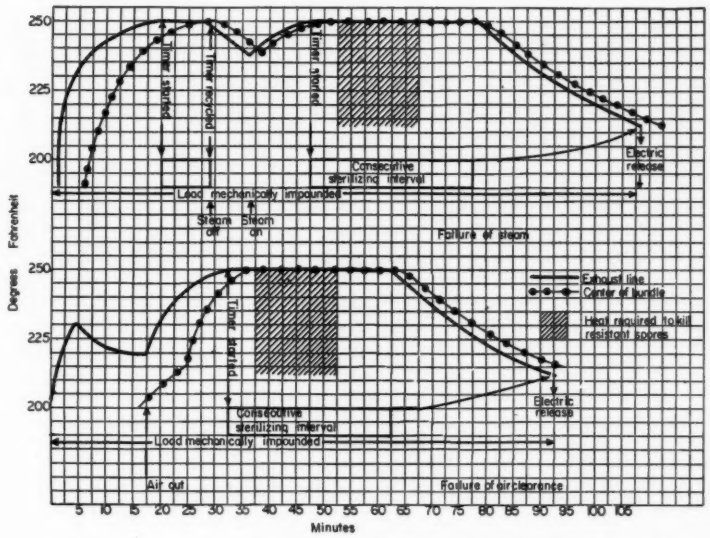


Fig. 6. Curves illustrating the action of the control under conditions of faulty sterilization. The load is not released until it has been sterilized.

9, 11, 19) and all of them disregard the human error which arises in their proper use (4, 16) and interpretation. None of the controls force the operator to correlate his interpretation of the detector with the actual operation of the sterilizer or the safe disposition of an unsterile load (1, 18), nor do they prevent unsterile supplies being removed from the poorly functioning or improperly operated sterilizer. To insure absolute sterility, a reliable control must not only reveal failure of the sterilizer itself, but must also prevent the human element from disregarding the warnings indicative of a dangerous fault in the process of sterilization.

A control (Fig. 3) has been designed and used in the Peter Bent Brigham Hospital for the past 3 years which *automatically impounds the load until it has been properly sterilized*, thus eliminating all opportunity for human error and insuring the absolute sterility of supplies. This control is fully automatic, indicates the various stages of the sterilizing cycle, and enforces a sequence of operation by impounding the load until the specified sterilizing cycle has been completed. The simplicity of design, rugged construction and housing of the control make it both foolproof and tamper-proof. The device is fail-safe because it acts to release the mechanical lock, which impounds the load, only after the satisfactory completion of a predetermined cycle. Thus failure of the control, whether mechanical or electrical, cannot result in the release of unsterile supplies. Any deviation from the established minimum of exposure to steam at sterilizing temperature is indicated immediately by appropriate signals; the control thus furnishes a guide for the proper operation of the sterilizer.

The autoclave door is locked by a rolling key clutch mechanism mounted within the handwheel assembly (Fig. 4). This clutch wedges the handwheel so that the locking bars cannot be retracted and simultaneously provides the takeup often necessary for sealing the door against leakage of steam as the chamber pressure builds up. After the satisfactory completion of the sterilizing cycle, the rolling keys are released from their wedging position by a magnetic trip.

The sterilizing cycle is checked by a recycling, synchronous electric timer which is controlled by sensitive thermoswitches located in the exhaust line of the sterilizer.

The sterilizer is operated according to the manufacturer's usual instructions. When the locking bars are forced into position to secure the sterilizer door, a switch is actuated which energizes the control. A red signal indicates that the sterilizer is locked but that the temperature is below that necessary for sterilization. After steam

has been admitted to the chamber and the temperature of the steam reaches the sterilizing level, a thermoswitch, set for 250 degrees F., starts the timer (Fig. 5). A combination of red and green signals now indicates that sterilizing conditions prevail. While the temperature of the steam remains above 250 degrees F., the timer meters a consecutive interval until 30 minutes have elapsed when it trips a switch which changes the signal to green alone indicating that the load is sterile and that the steam may be shut off and the vent valve opened.

After the pressure in the chamber has been relieved and the temperature falls to 210 degrees F., a second thermoswitch closes, releasing the clutch. A white signal indicates that the load is sterile and that the hand wheel may be turned to retract the locking bars.

If for any reason the temperature of the steam falls below 250 degrees F., the timer automatically recycles and the entire 30-minute period must be repeated (Fig. 6). Thus continuous exposure to saturated steam destructive to bacterial life is assured.

The use of this control provides a reliable check on sterilization in hospitals having dressing sterilizers of adequate size, because overloading of such sterilizers is unlikely.

In hospitals where the sterilizers are too small, overloading is almost the rule and further precautions must be taken to measure the penetration of steam into the bundles or drums. This can be done by wrapping a steam-proof thermoswitch, set for 250 degrees F., in the center of the largest bundle or most tightly packed drum. The timer then meters the duration of satisfactory sterilizing conditions in the center of the densest portion of the tightly packed load.

This automatic control provides the surgeon with a reliable method of eliminating faulty sterilization due to failure of the sterilizing equipment and ignorance or negligence on the part of the attendant. It enforces adherence to a predetermined standard of sterilization sufficient to insure absolute sterility of surgical supplies without waste of steam or damage to fabrics because of excessive exposure to steam.

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# EDITORIALS

SURGERY
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OCTOBER, 1938

## IMPROVEMENTS IN STERILIZING TECHNIQUE

ALTHOUGH every surgeon will agree that asepsis is the basis of his art, how many thoroughly understand how asepsis is attained? Are surgeons satisfied with the present methods for sterilization? The rapid development of surgery as a therapeutic measure has diminished the surgeon's interest in the preparation of the requisite instruments and supplies, and has focused his attention on the refinement of operative procedure and the exploration of new fields. In most hospitals, sterilization has become the responsibility of the chief nurse in the operating room. Problems which arise are referred either to the surgical staff or to the administration. Usually the nurses receive little expert advice, since neither the surgeon nor the administrator is equipped with sufficient bacteriological or engineering knowledge to be of real assistance. Thus the very foundation of safe surgical therapy lacks that attention and interest so essential to growth and perfection.

The failure of hospitals to appreciate the value of expert bacteriologists as staff members is partially responsible for such neglect. The appointment of bacteriologists to staff positions equal to that of the physician, surgeon, and pathologist will revive the lagging interest in this basic science. Then only will aseptic technique be studied critically. Meanwhile, epidemics of infected wounds and outbreaks of gas gangrene will occasion infrequent and often casual inspection or tests of the efficiency of the apparatus which makes our art possible.

A cursory survey of the sterilizing practices in hospitals throughout the country reveals striking differences and glaring inconsistencies not only in the technique used, but also in the sterilizing schedule of individual clinics. The principle of aseptic surgery—the reduction of the number of bacteria introduced into a surgical wound to a minimum—is so clear cut that such chaos leads one to suspect inefficiency and faulty sterilization. The concept of asepsis should permit the establishment of a standard of sterilization which would insure absolute sterility of all instruments and supplies used in the operative field. Expediency has forced surgeons to sanction questionable technique, because practical methods for absolute sterilization have not been available.

The emergency sterilization of instruments urgently needed during the course of an operative procedure causes many breaks in asepsis, because the accepted methods for "quick sterilization" are as faulty as they are quick. Sterile instruments can be provided in 3 minutes by using a rapid, high temperature auto-

clave constructed especially for the purpose.[1] Less efficient, unsafe methods have no place in the modern operating room.

The problem of cleansing and sterilizing instruments contaminated with virulent organisms and spores presents itself as frequently as the surgeon deals with sepsis or performs an intestinal operation. Instruments soiled with blood, pus, feces, or grease can be cleansed, sterilized, and dried rapidly and efficiently under superheated water in a new sterilizer[1], designed to remove the oils and protein residues during sterilization. This sterilizer can also be used to sterilize instruments before operation, thus assuring the surgeon of instruments free of spores.

The sterility of surgical dressings is often conceded merely because they have been through a pressure sterilizer. Various sterility detectors have been devised to assure the surgeon sterile supplies, but such detectors have proved inadequate. A new control,[2] which impounds dressings in the sterilizer until they have been exposed to saturated steam for a sufficient interval to destroy all bacterial life, can be used to modernize existing dressing sterilizers.

The improvements in mechanical appliances, outlined in the papers referred to, constitute advances in aseptic surgery of vital importance. The apparatus described has been evolved after careful study, its design is simple and sturdy, and its performance fulfills bacteriological requirements.

The small high temperature autoclave is essential in the operating room, if the surgeon and patient are to be spared unnecessary delay while waiting for the sterilization of an instrument indispensable for the completion of the operation. If rapid sterilization, such as that provided by this autoclave, is not available, then instruments are ordinarily inadequately sterilized. The use of superheated water for the sterilization of instruments, soiled with pus and feces, provides for safe terminal disinfection of potentially dangerous instruments.

Infection with spore bearing anaerobes constitutes one of the unpredictable hazards of surgery. If spores are protected by a surrounding layer of oil or grease in the locks of hemostats, sterilization by boiling is impossible and the chance of cross infection is enhanced. Thus, instruments from septic fields and intestinal cases should be cleaned and sterilized under conditions which prevent the dissemination of spores throughout the operating suite.

The new control for steam sterilization is an important advance, which insures sterile supplies by eliminating all opportunity for error due to mechanical failure or human negligence. Experience has shown it to be foolproof and reliable. Prolonged interest in the problem of sterilizing supplies and a realization of the many chances for error lead me to recommend immediate, widespread adoption of the new control.

Such advances in sterilizing technique, the emergency instrument sterilizer, the instrument washer and sterilizer, and the dressing sterilizer control, permit the establishment of a standard of sterilization heretofore but a theoretical attainment—the absolute sterility of all instruments and supplies reaching the operative field. ELLIOTT C. CUTLER.

[1]Walter, C. W. Technique for the rapid and absolute sterilization of nstruments. Surg., Gynec. & Obst., 1938, 67:244–248.
[2]Walter, C.W. A reliable control for steam sterilization. Surg., Gynec., & Obst., 1938, 67: 526–530.

## CANCER OF THE BREAST IN THE NEGRO

THE title is really a misnomer as we are not dealing with a pure race. It is generally agreed that cancer is rarely found in the pure native races which

have not been in contact with outside civilization. The negro was introduced into America in 1619 when slaves were first brought to Virginia. Between 1619 and 1790, 333,000 negroes were bought into bondage in the United States.

The negro population, according to the 1930 census, is 11,891,143, the white population at the same time being 108,864,207. The negro population represents definitely a mixed race. "For census purposes the term 'mulatto' includes all persons having some proportion or perceptible trace of negro blood." From 1850 to 1910 the proportion of mulattoes to the total negro population increased from 11.2 to 20.9 per cent. "The black population in 1910 was less than two and a half times as great as in 1850, while the mulatto population in 1910 was more than five times as great as in 1850."[1]

Education and living conditions for the negro, until recently, have not kept pace with the mixture of the races. Superstitions and fear, the natural associates of ignorance, have played a definite rôle in the development of the problems of today.

In 1930, according to the census, there were in the United States, 1,513,892 illiterate negroes. Ninety-three and six tenths per cent of these were in the South. In other words, about sixteen per cent of the negro population in 1930 were illiterates.

Education through medical centers is increasing the standards of practice among this group. It is important that stress be laid on the education of the profession. Dissemination of knowledge such as can be given to the laity is not sufficient because it does not reach all groups. Certainly, propaganda in the form of printed matter cannot have an effect upon the illiterate portion of the population. We must educate the profession at large so that the underprivileged groups will receive adequate examinations and advice. Nowhere is this more urgently needed than in the problem under consideration. This necessity can be best appreciated when certain facts are understood.

The mortality statistics collected by the Census Bureau indicate that in 1934 there were 13,171 deaths from cancer of the breast among the whole population. Of these there were only 779 among the colored population. The total negro population is 10 per cent of the entire population.

In centers where the negro population is high, there is a slight preponderance of cancer of the breast among negroes. In a recent study of 481 cases of cancer of the breast at Charity Hospital in New Orleans, 256 were negroes and 225 were white. There has not been a noticeable increase in the incidence of cancer of the breast among the negro race during the past five years. It has remained approximately 1.7 per thousand for total admissions during the five year period (1931–1936). Sixty per cent of these patients had metastases to the axilla and many of them already had skin and skeletal involvement when they came under observation. Less than 50 per cent of the cases observed are eligible for radical operation. The average duration of symptoms before medical care is sought is approximately 1.6 years, and more than 20 per cent of the patients seek advice after that period.

Pregnancy, cystic mastitis, and syphilis have apparently played a minor rôle as causative factors in cancer of the breast in the negro. Thirty-six of a total of 256 cases denied pregnancy at any time. From a series of 208 cases, clinical manifestations of syphilis were not obtainable in 187, and laboratory data were negative for syphilis in the 187 patients mentioned. There were only a few cases in which

[1]Negro Population (1790-1915), U. S. Department of Commerce. Statistics from *Negroes in the United States, 1920-1932*, from the United States Department of Commerce Bureau of the Census, published, 1935.

histological findings indicated the association of so called "cystic mastitis," with the cancer cases.

Cancer of the breast reaches its highest incidence in the decade 40 to 50, whereas in the white race, the maximum incidence is between 50 and 60.

Within a period of 1 to 4 years following operation for cancer of the breast, the known dead probably reaches the distressing figure of 75 per cent. This may be accounted for by the fact that cancer has been seen, as a rule, late in its course. Investigation of records indicates that adequate care is not always given to the study of the case before operation is resorted to. This does not apply only to cancer of the breast nor does it apply only to surgery in the negro race. It is equally true for all. The challenge to the profession is to educate themselves and to give adequate time to the study of each problem before unwise and unnecessary operations are performed.

After all, the differences between ability and usefulness of the surgeon lie not in the difference in knowledge but in the desire and willingness to give to each patient what is really needed.

Methods of treatment vary. The surgeon should use that form of treatment which his judgment, knowledge of the literature, experience, and ability fit him best to carry out. Some advise irradiation, x-ray or radium, either packs or interstitial implantation. It would seem justifiable to state that irradiation before operation is not indicated in any group of patients who present themselves at a late stage of the operable period. Valuable time is lost while waiting to complete the necessary period after irradiation. During this time, the radical operability of the patient may pass.

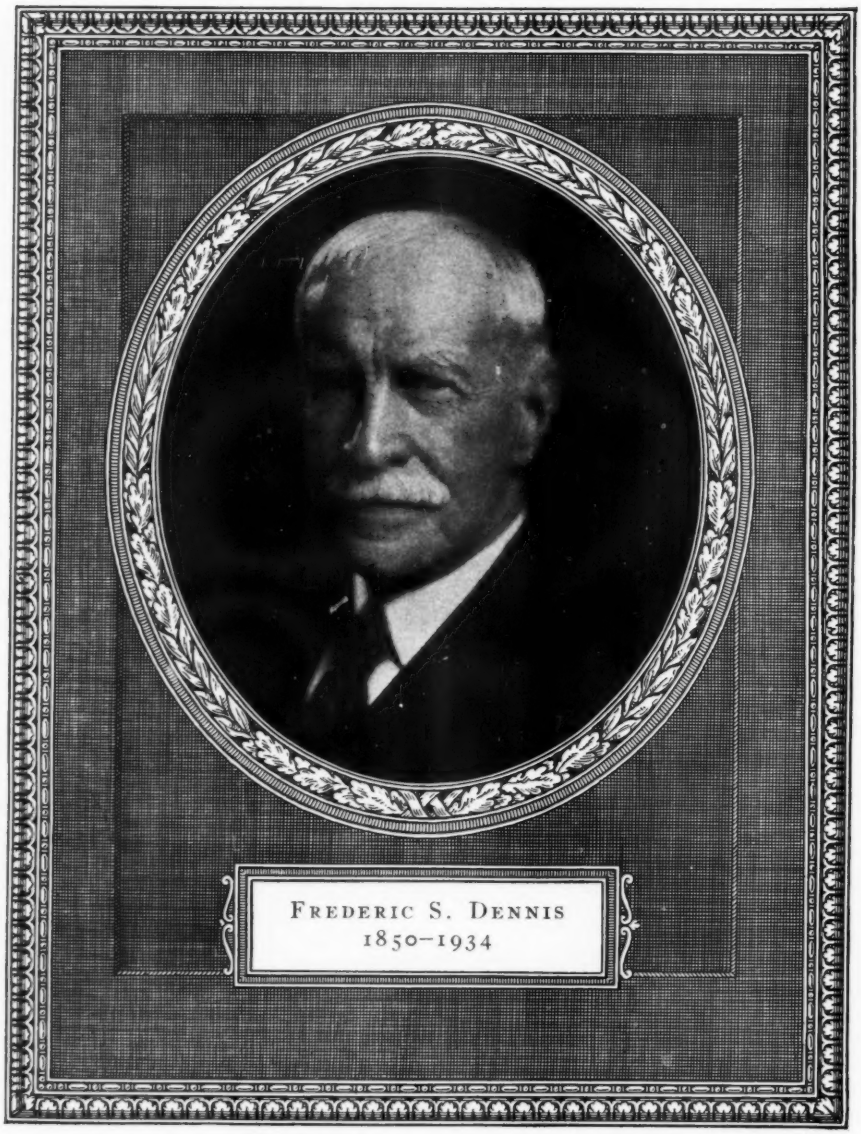
We have no positive proof that cancer of the breast is curable in a high percentage of cases by interstitial irradiation. The best available results are those of Mr. Geoffrey Keynes and his statements indicate that 50 per cent have evidence of active cancer cells in biopsies taken 6 months after interstitial irradiation.

Radical surgery when indicated offers the best opportunity for cure.

In order that better results may be obtained, a graduate educational program for the profession is essential. When the profession is more cancer conscious and, particularly, when they realize that cancer of the breast is not a rare condition among the negro population, they will examine their patients more adequately and they will also attempt to educate them.

Public education cannot adequately substitute for professional efficiency.

ISIDORE COHN



FREDERIC S. DENNIS
1850–1934

# MASTER SURGEONS OF AMERICA

## FREDERIC S. DENNIS

AT the close of the eighty-fourth year of his life, Frederic S. Dennis, of New York City, died at his home March 8, 1934.

To those of us who go back to the nineties and the earlier years of this century, Dr. Dennis was known as one of the leaders of the new thought in surgery which had been brought about by the work of Pasteur and Lister. He was not only an able surgeon, but his personal qualities were such as to win the warm friendship of all with whom he came in contact. His chief work in those earlier years, when I first knew him, was done at the Bellevue Hospital of New York, where his brilliant lectures and his skillful methods of operating won for him general admiration and respect.

Pasteur, a Frenchman, did more for the human race than any other man who ever lived, but French surgeons, while they accomplished much in the middle of the last century for fine anatomical dissections, had not fully accepted the relationship to disease as brought out by Pasteur's discoveries, just as the medical profession of Great Britain failed to grasp what Lister's discoveries meant to medicine and to surgery.

American surgeons of the earlier period, brilliant and well trained though they were in anatomy through the Scottish masters and in the gross pathology of the "dead-house" of the English tradition, were not as yet so versed in the "pathology of the living" as men who had been educated in Europe by the followers of Pasteur and Lister.

As I look back on this period in American surgery, I recall brilliant men whose names are familiar to the older members of the profession: in Boston, Richardson, Cheever, the Cabots, the Warrens, Watson, and many others; in New York, Dennis, Weir, McBurney, Bull, and others. In Philadelphia was that great triumvirate, the Grosses, father and son, and Agnew. And so one could go over the country, recounting great surgeons, trained anatomists, dead-house pathologists, but the majority of them at that time lacking in that essential thing which was brought in by Pasteur and Lister.

American students realized that what we had in America was the art of surgery based on the science of anatomy and dead-house pathology, but that we lacked the "living pathology" which was the foundation on which surgery must achieve its purpose.

In 1876 Dr. Dennis, in company with Dr. William H. Welch, his lifelong friend, went abroad, largely to study with Lister and gain an understanding of

those principles of surgical pathology, asepsis and antisepsis which the germ theory had brought forth. On his return, Dr. Dennis became one of the foremost teachers of the new era.

It would be idle for me to write of the many contributions Dr. Dennis made to surgical progress, by example illustrating the difference between the surgeon and the operator. Surgery is more a matter of mental grasp than it is of handicraftsmanship. I think all of us who have worked years in the profession understand that many very skillful operators are not good surgeons.

When one scans the years in which Dr. Dennis devoted himself not only to active surgical practice but to teaching surgery by the spoken word and by the written word, one is impressed by the wealth of his learned contributions to the literature. Among them was one of the greatest texts on surgery ever written, his *Systems of Surgery*, brought out in four volumes in 1895 and 1896.

Dr. Dennis for some years before his death reviewed his medical writings, with the idea of putting into book form certain selected surgical papers from this most valuable material which would represent the history of a surgical epoch. This work he completed.

I am grateful for this opportunity to express the appreciation of American surgeons for aid given by the highly educated, wise men of our profession, among whom none has been more esteemed than Frederic S. Dennis. He is held in honored memory.

W. J. MAYO.

# THE SURGEON'S LIBRARY

## REVIEWS OF NEW BOOKS

IN THE third edition of the popular work, *X-rays and Radium in the Treatment of Diseases of the Skin*[1] by MacKee are incorporated so many changes that it is really a new book. When the first edition was published almost 20 years ago, it was a pioneer effort on the part of the author but one that was most welcome to all dermatologists and all practitioners engaged in the treatment of skin diseases.

In the last few years there has been a remarkable increase in our knowledge of physics, biochemistry, physiology, histopathology, and the technique of administering x-rays and radium. Because of this the author has assigned the writing of many of these chapters to collaborators who are experts in their respective fields.

There are 42 chapters and among them are new chapters on physics, biology, histopathology, apparatus, and x-ray technique. The chapters on psoriasis, lichen planus, the hematopoietic diseases, fungus diseases, and pyodermas as well as the chapter dealing with miscellaneous diseases have all been revised and rewritten.

The author believes that there is an increasing indisposition on the part of dermatologists to employ x-rays only when necessary, and with discriminating judgment. In the clinical portions of the book devoted to the treatment of skin diseases, his own conservatism is reflected. These chapters should prove of inestimable value to the younger men.

The text is clear, concise and authoritative. There is an extensive bibliography at the end of each chapter. The paper is of excellent quality and the book is printed in large legible type. The illustrations are excellent.

The book embodies the latest established principles of roentgen therapy and their practical application and is most earnestly recommended to all practitioners interested in roentgen and radium therapy. It is the foremost authority in its field.

EDWARD A. OLIVER.

THE contributors to *Clinical Roentgen Therapy*[2] were: Chavany, Desjardins, Eller, Golden, Henderson, Holfelder, Hubeny, Juengling, Langer, MacKee, Martin, Meyer, Pohle, Richards, Sevringhaus, Waters, and Zimmern. Dedication honors the memory of the late Preston M. Hickey.

Preliminary chapters concern diseases of the blood and blood-forming organs. Some 20 pages are given over to the treatment of leucemia. There are chapters on diseases of the circulatory system; the respiratory system and the breast; some of the less frequently recognized indications in digestive disorders; more than 150 pages concerning radiation therapy in diseases of the female genital organs; and an additional 55 pages discussing similar diseases in the male. Further chapters cover the nervous system and the eye and ear. Chapter IX by Juengling is a notable contribution of over 100 pages on diseases of muscles, tendons, bones and joints, including fractures. Chapter X on the glands of internal secretion is a very interesting summary of the work in which Langer did much of the pioneering. Eller discusses diseases of the skin and MacKee reactions and injuries from the x-ray. An appendix is devoted to some of the medicolegal aspects of radiation therapy.

The book is authoritative, comprehensive, and gives us a very fair and reasonable presentation of the entire subject of radiation therapy. Here and there one might differ from the authors in minor details, but on the whole the book is a very dependable guide in the application of roentgen rays to the treatment of disease.

JAMES T. CASE.

THE appearance of the first edition of Kronfeld's *Introduction to Ophthalmology*[3] is most welcome. In the preface, the author states that "he has endeavored to formulate the principles underlying that portion of ophthalmology which is a necessary part of basic medical education. The presentation of these principles will, it is hoped, prove useful as a supplement to the short and chiefly practical courses to which the teaching of ophthalmology has had to be reduced in the curriculum of many medical schools. The material presented pertains principally to the pathogenesis of disease. The details of diagnosis, of methods of examination, and of treatment have been omitted. Individual diseases are discussed in this book to illustrate pathogenetic principles. A large number of common diseases have been omitted altogether from the text because they are unimportant for the exposition of ophthalmological principles. Of these diseases the reader will find short descriptions in the dictionary which is combined with the index of this book."

The text is by no means elementary. In this respect it cannot be intended for undergraduate students alone; it can, however, be recommended highly for internes and residents specializing in

[1] X-RAYS AND RADIUM IN THE TREATMENT OF DISEASES OF THE SKIN. By George M. MacKee, M.D. 3d rev. ed. Philadelphia: Lea & Febiger, 1938.

[2] CLINICAL ROENTGEN THERAPY. Edited by Ernst A. Pohle, M.D., Ph.D., F.A.C.R. Foreword by George W. Holmes, M.D. Philadelphia: Lea & Febiger, 1938.

[3] INTRODUCTION TO OPHTHALMOLOGY. By Peter C. Kronfeld, M.D. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1937.

ophthalmology and for practicing oculists. The current thought in ophthalmology is presented clearly and concisely, especially in the chapters on vascular diseases of the eye and in chapter on visual pathway. It is obvious that the author's extensive teaching experience is exemplified in these chapters.

The subject matter is presented with simplicity and clarity in those problems in which the young oculist can be most easily confused if he resorts merely to a perusal of the current literature.

The binding, paper, and print are admirable, and an excellent index and ophthalmological dictionary close the work. The illustrations, although limited as to number, are excellent and really illustrative. This book is another example of the excellent texts on ophthalmology that are being produced in this country which are freeing the American ophthalmologists from the nearly century old bonds of Teutonic domination of the subject.

HARRY S. GRADLE.

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## BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

DER ZYKLUS DER FRAU; REFORM DES EHELEBENS. By Dr. Jules Samuels. The Hague: G. Naeff, 1938.

FRACTURES OF THE JAWS. By Robert H. Ivy, M.D., D.D.S., F.A.C.S., and Lawrence Curtis, A.B., M.D., D.D.S., F.A.C.S. 2d rev. ed. Philadelphia: Lea & Febiger, 1938.

THE PRINCIPLES AND PRACTICE OF MEDICINE; DESIGNED FOR THE USE OF PRACTITIONERS AND STUDENTS OF MEDICINE. Originally written by the late Sir William Osler, Bart., M.D., F.R.C.P., F.R.S. Revised by Henry A. Christian, M.D., LL.D., S.D., F.R.C.P. The 9th, 10th, 11th and 12th eds. revised by Thomas McCrae, M.D., F.R.C.P. 13th ed. New York and London: D. Appleton-Century Co. Inc., 1938.

ANUS, RECTUM, SIGMOID COLON; DIAGNOSIS AND TREATMENT. By Harry Ellicott Bacon, B.S., M.D., F.A.C.S., F.A.P.S. Introduction by W. Wayne Babcock, A.M., M.D., LL.D., F.A.C.S. Foreword by J. P. Lockhart-Mummery, M.A., M.B., B.C.(Cantab.), F.R.C.S.(Eng.). Philadelphia, Montreal, and London: J. B. Lippincott Co., 1938.

LIFE AND LETTERS OF FIELDING H. GARRISON. By Solomon R. Kagan, M.D. With an introduction by Prof. James J. Walsh. Boston, Mass.: The Medico-Historical Press, 1938.

APUNTES DE TÉCNICA OPERATORIA (Temas de práctica diaria). Primer cuaderno pre y post-operatorio. By Dr. Ivan G. Moreno. Buenos Aires: Libreria y Editorial "El Ateneo," 1938.

THE PRINCIPLES AND PRACTICE OF MEDICAL NURSING. By Victor Knapp, M.D. New York: G. P. Putnam's Sons, 1938.

APPLIED ANATOMY; FUNCTIONAL AND TOPOGRAPHICAL. By Robert H. Miller, M.D. Philadelphia: Lea & Febiger, 1938.

EXPERIENCE IN THE MANAGEMENT OF FRACTURES AND DISLOCATIONS (Based on an Analysis of 4390 Cases). By The Staff of the Fracture Service, Massachusetts General Hospital, Boston; under the general editorship of Philip D. Wilson, M.D. Philadelphia, London, and Montreal: J. B. Lippincott Co., 1938.

# CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

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## PROGRAM FOR THE 1938 CLINICAL CONGRESS

THE complete program for the twenty-eighth annual Clinical Congress of the American College of Surgeons, to be held in New York and Brooklyn, October 17 to 21, appears in the following pages.

Under the leadership of strong and representative committees, the surgeons of greater New York have prepared a program of operative clinics and demonstrations that will provide a complete showing of their clinical activities in all departments of surgery. The Committee on Arrangements has the hearty co-operation of the clinicians at the five medical schools and more than sixty hospitals that will participate in the clinical program. Clinics will be held on the afternoon of Monday, October 17, and on the mornings and afternoons of each of the following four days. Wednesday, October 19, has been designated as Brooklyn-Long Island Day, and the clinical program on that day will be presented in Brooklyn hospitals.

It will be noted that the program provides an ample and well arranged schedule of operative clinics, at which the technique of a wide variety of surgical procedures will be demonstrated in the operating rooms, and in addition, the committees have arranged a series of non-operative clinics in many of the large hospitals and medical schools for the presentation of the work being done in many special fields. The programs are so correlated that the visiting surgeon may devote his time continuously, if he so desires, to clinics dealing with the special subject in which he is most interested. For example, clinics in fractures, neurosurgery, thoracic surgery, plastic surgery, cancer, etc., will be available each forenoon and afternoon during the Congress.

The actual program of the Congress will be published from day to day in the *Daily Clinical Bulletin.* Each afternoon the complete detailed clinical program for the following day will be posted in the form of bulletins at headquarters. The same material will be published in printed form for distribution the following morning.

The annual meeting of the governors and fellows of the College will be held in the ballroom of the Waldorf-Astoria on Thursday afternoon at 1:30 o'clock. Reports on activities of the College

### COMMITTEE ON ARRANGEMENTS

#### NEW YORK

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HOWARD A. PATTERSON, Secretary
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FREDERIC W. BANCROFT
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ALLEN O. WHIPPLE
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#### BROOKLYN-LONG ISLAND

DONALD E. MCKENNA, Chairman
CHARLES A. ANDERSON
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CLINICAL CONGRESS PROGRAM IN BRIEF

All sessions at the Waldorf-Astoria except as noted.

*Monday, October 17*

10:00 Hospital Conference—Ballroom.
2:00 Clinics in New York Hospitals.
2:00 Hospital Conference—Sert Room.
2:00 Surgical Film Exhibition—Empire Room.
3:00 Meeting of Initiates—Ballroom.
4:00 Reception to Fellows and Initiates—Ballroom.
8:00 Presidential Meeting and Convocation—Ballroom.

*Tuesday, October 18*

9:00 Clinics in New York Hospitals.
9:30 Hospital Conference—Sert Room.
9:30 State and Provincial Judiciary Committees—Empire Room.
10:00 State and Provincial Credentials Committees—Empire Room.
11:00 State and Provincial Executive Committees—Empire Room.
12:00 Clinical Conferences—Jansen Suite, Blue Room, Assembly and Carpenter Suites.
2:00 Clinics in New York Hospitals.
2:00 Cancer Symposium—Ballroom.
2:00 Hospital Conference—Sert Room.
2:00 Surgical Film Exhibition—Empire Room.
8:00 Scientific Session, General Surgery—Ballroom.
8:00 Scientific Session, Ophthalmology—Sert Room.
8:00 Scientific Session, Otorhinolaryngology—Empire Room.
8:00 Hospital Conference—Jansen Suite.

*Wednesday, October 19—Brooklyn Day*

9:00 Clinics in Brooklyn Hospitals.
10:00 Hospital Conference—Sert Room.
10:00 Surgical Film Exhibition—Ballroom.
12:00 Clinical Conferences—Jansen and Assembly Suites Blue Room, Empire Room.
2:00 Scientific Session, General Surgery—Ballroom.
2:00 Hospital Demonstrations—New York Hospitals.
2:00 Clinics in Brooklyn Hospitals.
8:00 Scientific Session, General Surgery—Ballroom.
8:00 Surgical Film Exhibition (Ophthalmology and Otolaryngology)—Empire Room.

*Thursday, October 20*

9:00 Clinics in New York Hospitals.
10:00 Hospital Conference—Sert Room.
10:00 Surgical Film Exhibition—Empire Room.
1:30 Annual Meeting—Ballroom.
2:00 Clinics in New York Hospitals.
2:00 Hospital Conferences—Sert Room and LePerroquet Suite.
3:00 Symposium on Industrial Medicine and Traumatic Surgery—Ballroom.
3:00 Surgical Film Exhibition—Empire Room.
8:00 Scientific Session, General Surgery—Ballroom.
8:00 Scientific Session, Otorhinolaryngology—Empire Room.
8:00 Scientific Session, Ophthalmology—Sert Room.

*Friday, October 21*

9:00 Clinics in New York Hospitals.
10:00 Surg.cal Film Exhibition—Empire Room.
12:00 Clinical Conferences—Jansen, LePerroquet and Assembly Suites, Blue Room.
2:00 Clinics in New York Hospitals.
2:00 Fracture Symposium—Ballroom.
2:00 Symposium on Obstetrics and Gynecology—Sert Room.
2:00 Urological Symposium—LePerroquet Suite.
2:00 Surgical Film Exhibition—Empire Room.
8:00 Community Health Meeting—Ballroom.

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will be presented by the officers and chairmen of standing committees, to be followed by the election of officers.

The attention of fellows is called to the meetings of three committees to be held in the Empire Room on Tuesday forenoon, as follows: State and Provincial Judiciary Committees at 9:30, State and Provincial Credentials Committees at 10, State and Provincial Executive Committees at 11.

The showing of surgical motion picture films, which so faithfully depict clinical features of major interest to most surgeons, will be continued at this year's Congress. It is planned to present an enlarged program of both sound and silent pictures at daily exhibitions at headquarters.

### PRESIDENTIAL MEETING AND CONVOCATION

The combined presidential meeting and convocation will be held in the ballroom of the Waldorf-Astoria on Monday evening at 8 o'clock, opening with a processional of the officers, regents, and honorary guests. Following the invocation, Dr. Henry W. Cave, Chairman of the Committee on Arrangements, will deliver the address of welcome, and the foreign guests will be introduced. Dr. Frederic A. Besley, of Waukegan, the retiring president of the College, will deliver the presidential address, after which the new officers will be inaugurated and fellowships conferred upon the initiates. Honorary fellowships will then be conferred and the medical records honor list and prize award announced.

The concluding feature of the program will be the annual oration on surgery, to be delivered by Dr. Walter W. Chipman, of Montreal, a former president of the College, whose subject will be "Our College Mandate—A Tribute to Allen B. Kanavel."

### ASSEMBLY OF INITIATES

Following the plan adopted last year, all of the convocation ceremonies, except the formal conferring of fellowship, will be included in a session on Monday afternoon preceding the combined convocation and presidential meeting in the evening. The afternoon assembly will be held in the ballroom of the Waldorf-Astoria and will be attended by the initiates and fellows

wearing the fellowship gown. Officials of the College will interpret the program of the College and the president-elect will extend official greetings. The initiates will recite the fellowship pledge and sign the fellowship roll, after which a reception will be held by the officers and regents for the Fellows and initiates and members of their families.

### SCIENTIFIC SESSIONS

In the general scientific sessions scheduled for Tuesday, Wednesday, and Thursday evenings, the aim in many of the subjects to be covered is to correlate the medical and the surgical phases of treatment of various specific pathological conditions. Several different subjects will be presented each evening. The Brooklyn Chapter of the College has co-operated in arranging the Wednesday evening program.

Sessions on ophthalmology and on otorhinolaryngology will be held on Tuesday and Thursday evenings. A feature of the Tuesday evening sessions, in addition to the scientific papers presented, will be discussions of graduate training for these specialties.

The afternoon sessions will include symposia on the subjects of cancer, industrial medicine and traumatic surgery, and fractures, scheduled for Tuesday, Thursday, and Friday respectively. On Friday afternoon there will be two additional symposia—one on obstetrics and gynecology, and the other on urologic infections. The session to be held on Wednesday afternoon will be devoted to a symposium on the subject of "Surgical Procedures on the Handicapped Patient," in which anesthetists and internists as well as surgeons will participate.

The midday round table conferences on Tuesday, Wednesday, and Friday, are a new feature of the Congress. There will be twelve in all, each devoted to a specific subject so restricted as to be more suitably covered in this way than in the general session. The leader will present the subject and selected authorities will discuss the various phases briefly. Then an open forum, directed by the leader and collaborators, will be devoted to the presentation of ideas informally. Since the rooms in which these discussions will be held are not large, registration will be limited by their capacity and should therefore be made as early as possible.

All of the programs, details of which are presented in the following pages, have been arranged by the program committee to include material of the widest possible interest, with consideration of the viewpoints of the specialist as well as of the general surgeon.

### BROOKLYN–LONG ISLAND DAY

A revised program for Brooklyn-Long Island Day (Wednesday) has been effected which promises to serve the best interests of the visiting fellows. The program will be concentrated in eleven institutions located in mid-Brooklyn which afford the physical accommodations required. The institutions selected are of such size as to provide comfortably for all of the visiting fellows, and their geographic location is such that the distance from headquarters is but a matter of minutes.

Through the courtesy of the Brooklyn and Long Island chapter, free transportation from the Waldorf-Astoria to the Brooklyn institutions via taxicab has been arranged, effective between the hours of 8 and 10 a.m. This will obviate the necessity of depending upon the usual transportation and will assure prompt arrival of the fellows at their desired destinations.

At each institution a morning program of both operative and dry clinics will be presented from 9 a.m. to 12:30 p.m., followed by a luncheon to the visitors. The afternoon has been set aside for the presentation of symposia covering all of the specialties and arrangements have been made for adequate transportation from one hospital to another at midday, so as to facilitate attendance at any symposium selected. Each symposium will be presided over by a moderator, whose duty it will be to keep the papers within the time limit, so that the programs will conclude at 4 p.m. The evening session will be held in the ballroom of the Waldorf-Astoria at 8 o'clock.

### GRADUATE TRAINING FOR SURGERY

The American College of Surgeons was founded on the determination to raise the standards of surgery. That its concept of those standards has developed with the progress of the art and science of surgery is proved by the insistence of the College upon systematic supervision of the young surgeon's first few years of surgical practice. The College program for increasing the opportunities for graduate training in general surgery and the surgical specialties, adopted after careful study of present plans and the needs of the immediate future, has reached the stage of practical application. The symposium in which this subject will be covered should claim the attendance of every fellow and of every hospital executive, because it will picture the progress and the prospects of one of the most important movements to stimulate better surgery.

This symposium on graduate training for surgery and the surgical specialties will be held at the opening session of the Hospital Conference in the

Waldorf-Astoria ballroom on Monday, at 10 a.m. The committee will present its second annual report through its chairman, Dr. Dallas B. Phemister of Chicago. This will be based on personal surveys by representatives of the College and the data contributed by more than three hundred hospitals which are or could readily be organized and equipped to offer acceptable graduate training.

Following presentation of the report, a member of the survey staff will offer practical suggestions for organizing and executing plans for graduate training. Then, in a panel discussion, will be given the ideas on content of courses for adequate training from the viewpoints of the general surgeon, the obstetrician and gynecologist, the neurosurgeon, the urologist, and the orthopedist. This will be supplemented by general discussion.

The plan of the College for graduate training, it should be repeated, is no longer in the formative stage—it is ready for practical application and has already been adopted by many institutions. To be conversant with its full implications from an educational and practical viewpoint is to understand one of the most potent influences operating to affect the future of surgery, since necessarily scientific progress will be promoted by raising training standards. Every approved hospital large enough to offer the diversification of practice and the facilities for graduate training, either directly or through co-operation with a medical school, should become familiar with the requirements, because it is desirable that guided instruction in surgery shall become a universal practice in order to protect the patient from the inexperienced surgeon. This symposium should be recognized as one not to be omitted from the schedule of any one wishing to keep informed on current developments in surgery.

### HOSPITAL CONFERENCE

Official announcement of the 1938 list of approved hospitals will be made following the address of the president of the College at the opening session of the twenty-first annual hospital standardization conference in the ballroom of the Waldorf-Astoria on Monday morning, at 10 o'clock. Thereafter a report of the survey on graduate training for surgery (general surgery and the surgical specialties) will be presented and the remainder of that session of the hospital conference will be devoted to a discussion of what constitutes adequate training in general surgery and the surgical specialties and how this program can be put into effect in selected hospitals.

The important subject of co-operation between voluntary and governmental or tax-supported hospitals will be discussed by the first speaker at the Monday afternoon session, to be held in the Sert Room of the hotel. This will be followed by two papers in which important nursing problems will be considered. Additional subjects will be personnel management, the organization and management of volunteer service in hospitals, and in conclusion a new subject which is now being accorded a scientific cognomen when used in the care of the patient—bibliotherapy.

Physical factors affecting patients and personnel will be the chief topic of discussion at the Tuesday morning session at 9:30 in the Sert Room. Light, noise, air conditioning, provisions for isolation of certain patients, and preparedness for emergencies will be considered. At this session will also be given a paper on sources and control of infections. The final feature will be a talk illustrated by motion pictures on blood bank service.

"The Care of the Mother and the Newborn in the General Hospital" will be the subject of the panel discussion scheduled for Tuesday afternoon, beginning at 2 o'clock. Dr. Malcolm T. MacEachern, associate director of the College, will present the minimum requirements of the College for an obstetrical department in a general hospital, followed by discussion from various viewpoints, and concluding with a presentation of the New York-Brooklyn plan of evaluation of maternal fatalities as conducted by the Committee of the New York and King County Medical Societies.

On Wednesday morning, at an important joint conference with the Association of Record Librarians of North America, the papers and discussions will embrace problems of procuring, compiling, and using medical records. This association is working to solve many of the practical problems involved in keeping adequate medical records in hospitals.

Wednesday afternoon the scene will shift to local hospitals where demonstrations of administrative and technical procedures of general and specialized interest to hospital executives and personnel will be conducted.

Thursday morning, in the Sert Room, a symposium on the all-important subject of the training of hospital executives will be presented, with discussion from a number of viewpoints of the need for such training. At the closing session of the conference on Thursday afternoon an administrative panel round table conference will be conducted by Robert Jolly of Houston, Texas, in which a great many questions and problems, submitted beforehand or during the session, will be answered and discussed by selected authorities or by the general assembly.

### MEETING ON HEALTH CONSERVATION

A community health meeting, as in former years a part of the Clinical Congress, is scheduled for Friday evening in the ballroom of the Waldorf-Astoria. The program, with the general title of "Health Conservation—Our Mutual Contributions," will consist of several short talks, most of them illustrated, on the activities of the College, scientific medicine, hospitals and health—aimed to give authoritative information to the interested layman and to acknowledge the contributions of the public to the advancement of the science of medicine, which have established a mutually helpful relationship between the public and the profession. Dr. Howard C. Naffziger, president of the College, will preside. The program follows:

The American College of Surgeons—Its Purposes—Its Program. George Crile, M.D., Cleveland.

Seven Wonders of Medicine (Illustrated). Bowman C. Crowell, M.D., Chicago.

What Is an Approved Hospital? (Illustrated). Malcolm T. MacEachern, M.D., Chicago.

Medical Science and the Public. Irvin Abell, M.D., Louisville.

Man, Research, and Disease. Clarence Cook Little, Sc.D., Bar Harbor, Maine.

Prevention of Disease. Frank H. Lahey, M.D., Boston.

The Voluntary Hospital. Walter W. Chipman, M.D., Montreal.

### HEADQUARTERS—TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Waldorf-Astoria Hotel, on Park Avenue between 49th and 50th Streets, where the grand ballroom and large adjacent foyers, the Astor Gallery, Jade and Basildon Rooms, all on the third floor of the hotel, have been reserved for Congress headquarters—scientific sessions and conferences, and for the scientific and technical exhibits.

The technical exhibition, together with the registration and clinic ticket bureaus, will be located in the East Foyer, Astor Gallery, Jade and Basildon Rooms, all on the third floor of the hotel. The bulletin boards, on which the daily clinical program will be posted each afternoon for the following day, will be placed in these rooms. Leading manufacturers of surgical instruments, x-ray apparatus, sterilizers, operating room lights, ligatures, dressings, hospital apparatus and supplies of all kinds, pharmaceuticals and publishers of medical books will be represented in this exhibition.

### NEW YORK HOTELS AND THEIR RATES

In addition to the headquarters hotel, the Waldorf-Astoria, there are many first-class hotels within short walking distance of headquarters, providing ample hotel facilities at reasonable rates. It is suggested that reservation of hotel accommodations be made at an early date. The following hotels are recommended by the Committee:

| | Minimum Rate with Bath | |
|---|---|---|
| | Single | Double |
| Ambassador, Park Ave. at 51st St. | $6.00 | $8.00 |
| Barclay, 111 East 48th St. | 6.00 | 10.00 |
| Belmont-Plaza, Lexington Ave. at 49th St. | 3.50 | 6.00 |
| Biltmore, Madison Ave. at 44th St. | 6.00 | 8.00 |
| Chatham, Vanderbilt Ave. at 48th St. | 4.00 | 7.00 |
| Commodore, 42nd St. at Lexington Ave. | 3.50 | 5.00 |
| Lexington, Lexington Ave. at 48th St. | 3.50 | 4.50 |
| New Weston, Madison Ave. at 50th St. | 4.00 | 6.00 |
| Park Lane, 299 Park Ave. | 6.00 | 8.00 |
| Ritz-Carlton, Madison Ave. at 46th St. | 7.00 | 9.00 |
| Roosevelt, Madison Ave. at 45th St. | 5.00 | 7.00 |
| Shelton, Lexington Ave. at 48th St. | 3.00 | 4.50 |
| Waldorf-Astoria, Park Ave. at 50th St. | 7.00 | 10.00 |

### ADVANCE REGISTRATION

The hospitals and medical schools of Greater New York afford accommodation for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to a number that can be comfortably accommodated at the clinics. The limit of attendance will be based upon the result of a survey of the operating rooms and laboratories of the hospitals and medical schools, to determine their capacity for visitors. It is expected, therefore, that those surgeons who wish to attend the Congress will register in advance. A registration fee will be required of surgeons attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the Congress. To each surgeon registering in advance a formal receipt will be issued, which is to be exchanged for a general admission card upon his registration at headquarters during the Congress. This card is not transferable and must be presented in order to secure clinic tickets and admission to scientific sessions.

A resolution adopted by the Board of Regents provides that the registration fee for fellows and endorsed junior candidates shall be $5.00; that no fee for the 1938 Congress shall be required of initiates (class of 1938); that the fee for nonfellows attending as invited guests of the College shall be $10.00.

Admittance to clinics and demonstrations at the hospitals will be controlled by means of clinic tickets. This plan provides an efficient means for the distribution of the visiting surgeons among the various clinics and assures against overcrowding. The number of tickets issued for any clinic

will be limited to the capacity of the room in which the clinic is given.

RAILROAD FARES

No special rates have been authorized by the railroads for the 1938 Clinical Congress in New York and Brooklyn so that certificates will not be required. However, the railroads in the western, northwestern and southwestern states will offer for sale in October round-trip tickets to New York, via the Chicago and St. Louis gateways, with a 30-day return limit. In the southern states round trip tickets will also be available.

In the territory east of Chicago and St. Louis, north of the Ohio and Potomac rivers, including the north Atlantic and New England states and the eastern provinces of Canada, regular rates of 3 cents per mile in pullmans and 2½ cents per mile in coaches will be in effect.

Local ticket agents will supply complete information as to rates, routes and stop-over privileges.

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# ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

*Monday, 10:00—Ballroom, Waldorf-Astoria Hotel*

FREDERIC A. BESLEY, M.D., Waukegan, President, American College of Surgeons, presiding.

Address of the President—Twenty-one Years of Hospital Standardization; Resulting Benefits to Medical Science.

The 1938 Hospital Standardization Survey—Official Announcement of the List of Approved Hospitals. GEORGE CRILE, M.D., Cleveland, Chairman, Board of Regents, American College of Surgeons.

Report of Survey—Graduate Training for Surgery and the Surgical Specialties. DALLAS B. PHEMISTER, M.D., Chicago.

Organizing and Executing a Plan for Graduate Training for Surgery in a Hospital. HAROLD EARNHEART, M.D., Chicago.

Panel Discussion—Content of Courses for Adequate Training in General Surgery and the Surgical Specialties, from the following viewpoints:

General Surgery. ALLEN O. WHIPPLE, M.D., New York.

Obstetrics and Gynecology. JOHN R. FRASER, M.D., Montreal.

Neurosurgery. HOWARD C. NAFFZIGER, M.D., San Francisco.

Urology. HERMAN L. KRETSCHMER, M.D., Chicago.

Orthopedics. PHILIP D. WILSON, M.D., New York.

Discussion. Led by ARTHUR M. WRIGHT, M.D., New York, and DERYL HART, M.D., Durham, N. C.

*Monday, 2:00—Sert Room, Waldorf-Astoria Hotel*

ALLAN CRAIG, M.D., Bangor, Maine, presiding.

Co-operation Between Voluntary and Governmental or Tax-Supported Hospitals. S. S. GOLDWATER, M.D., New York.

Present Trends in Nursing as Affecting Nursing Education and Nursing Service in Hospitals. EFFIE J. TAYLOR, New Haven.

A Grading Program for Schools of Nurses. REV. A. M. SCHWITALLA, S.J., St. Louis.

Personnel Management. JOSEPH C. DOANE, M.D., Philadelphia.

The Organization and Management of Volunteer Service in the Hospital. CHRISTOPHER G. PARNALL, M.D., Rochester, N. Y.

The Rôle of Bibliotherapy in the Care of the Patient. GORDON R. KAMMAN, M.D., St. Paul.

Discussion. Led by OLIVER H. BARTINE, Bridgeport.

*Tuesday, 9:30—Sert Room, Waldorf-Astoria Hotel*

FRASER D. MOONEY, M.D., Buffalo, presiding.

Panel Round Table Discussion—Physical and Other Conditions in the Hospital Related to the Care of the Patient and the Working Conditions of the Personnel:

Lighting in the Operating Room. WILLIAM J. ENGEL, M.D., Cleveland.

Air Conditioning in Hospitals. VICTOR A. FRID, Hartford, Conn.

Noise in the Hospital; Its Effect on Patients; Its Control. HARVEY AGNEW, M.D., Toronto.

Emergency Lighting in Hospitals. CHARLES F. NEERGAARD, New York.

Provision for Isolation of Infected Patients in General Hospitals. A. J. MCRAE, M.D., Hempstead, N. Y.

Preparedness for Emergencies. MIRIAM CURTIS, R.N., Northampton, Mass.

Infections; Sources and Control. CLAUDE W. MUNGER, M.D., New York.

Blood Bank Service (Illustrated by motion picture). KARL A. MEYER, M.D., LEONARD H. WEISSMAN, M.D., and J. LESTER WILKEY, M.D., Chicago.

*Tuesday, 2:00—Sert Room, Waldorf-Astoria Hotel*

GEORGE W. KOSMAK, M.D., New York, presiding.

Presentation of the Minimum Requirements of the American College of Surgeons for the Obstetrical Department in a General Hospital. MALCOLM T. MACEACHERN, M.D., Chicago.

Panel Discussion—The Care of the Mother and Newborn in the General Hospital.

Discussion from the viewpoints of:

Organization of the Obstetrical Department so as to Provide Administrative and Clinical Efficiency and Control. FRED L. ADAIR, M.D., Chicago.

Provision and Indications for Segregation and Isolation of Obstetrical Patients and Newborn to Prevent Infection. HERMAN W. JOHNSON, M.D., Houston, Texas.

Organization of the Obstetrical Staff with Particular Reference to Qualifications and Grading of Privileges. HARVEY B. MATTHEWS, M.D., Brooklyn.

Indications for Consultations on the Obstetrical Service and Proper Procedure in Securing These. PAUL TITUS, M.D., Pittsburgh.

Analysis of the Clinical Work of the Obstetrical Service with Special Emphasis on Morbidities and Mortalities. JAMES R. MILLER, M.D., Hartford.

Assuring the Mother and Newborn Efficient Nursing Care. JESSIE J. TURNBULL, R.N., Pittsburgh.

Proper Training of Interns and Residents in Obstetrics through Arrangement of Services, Supervision of Work, and Instruction. SAMUEL A. COSGROVE, M.D., Jersey City.

Palm Print Method of Infant Identification with Demonstration. GILBERT P. POND, M.D., Oak Park, Ill.

An Evaluation of Maternal Deaths (Outline of procedure developed by the Committees on Maternal Welfare of the medical societies of the counties of New York and Kings, for judging the responsibility in each case of pregnancy fatality). GEORGE W. KOSMAK, M.D., ALFRED M. HELLMAN, M.D., New York, and CHARLES A. GORDON, M.D., Brooklyn.

*Tuesday, 8:00 p.m.—Jansen Suite, Waldorf-Astoria Hotel*

Joint Session with the Greater New York Hospital Association.

CLAUDE W. MUNGER, M.D., New York, presiding.

The Place of the Voluntary Hospital in Society. DAVID H. MCALPIN PYLE, New York.

The Safeguarding of Trust Funds. WILLIAM H. WALSH, M.D., Chicago.

The Advantages of Simplification and Standardization of Hospital Furnishings, Equipment, and Supplies. L. M. ARROWSMITH, Brooklyn.

Trends in Hospital Insurance. C. RUFUS ROREM, Ph.D., Chicago.

The Rôle of the Hospital in Graduate Medical Education. R. C. BUERKI, M.D., Madison, Wis.

*Wednesday, 9:30—Sert Room, Waldorf-Astoria Hotel*

Joint Conference with Association of Record Librarians of North America.

JAMES T. NIX, M.D., New Orleans, presiding.

The Program of the Association of Record Librarians of North America as it Affects Hospitals. JENNIE C. JONES, R.R.L., Baltimore.

The Ills of Medical Records and Their Remedies: Delay in Writing, Incompleteness, Unscientific Value, Insufficient Use. GORDON R. KAMMAN, M.D., St. Paul.

The Medical Records Librarian: Qualifications, Responsibilities, and Duties. HELEN ROBINSON, R.R.L., Little Rock, Ark.

How the Medical Records Librarian Can Assist the Physician in Securing Medical Records. NORMA SWANSON, Red Wing, Minn.

The New York Hospital Classified Nomenclature of Operations. BRONSON S. RAY, M.D., New York.

Panel Discussion—Uses of the Medical Record:

Monthly Analysis Report for Medical Audit. LEONARD SHAW, Chicago.

Making Group Studies of Diseases. MARY M. NEWTON, B.A., R.N., Peoria, Illinois.

Preparing Scientific Papers. ALFRED W. ADSON, M.D., Rochester.

Clinical Research. FRANK E. ADAIR, M.D., New York.

Discussion. Led by JOSEPH R. CLEMMONS, M.D., New York.

*Wednesday, 2:00—Local Hospitals*

Demonstrations of Administrative and Technical Procedures in Local Hospitals:

Anesthesia. New York Post-Graduate Medical School and Hospital.

The Care of Chronic Patients. Montefiore Hospital.

Central Record Room and Follow-up System. Mount Sinai Hospital.

Clinic Management. Vanderbilt Clinic.

Food Service. New York Hospital.

Isolation Technique. Willard Parker Hospital.

Medical Social Service. St. Luke's Hospital.

Nursing Service. Queens General Hospital, Jamaica.

Oxygen Therapy. Presbyterian Hospital.

Simplified Economical Method of Preparing Sterile and Parenteral Solutions. Roosevelt Hospital.

*Thursday, 9:30—Sert Room, Waldorf-Astoria Hotel*

R. C. BUERKI, M.D., Madison, presiding.

Symposium: The Training of Hospital Executives.

Need for Adequate Education and Training for Hospital Executives. JAMES A. HAMILTON, New Haven.

Discussion from the following viewpoints:

Apprenticeship in Hospital Administration. DONALD C. SMELZER, M.D., Philadelphia.

Graduate and Undergraduate University Courses for Hospital Administrators. GERHARD HARTMAN, Chicago.

Institutes for Hospital Administrators. NEAL N. WOOD, M.D., Chicago.

Supplementary Training and Experience in Hospital Administration—Reading, Observation Tours, and Attendance at Meetings. GEORGE A. MACIVER, M.D., Worcester, Mass.

Discussion. Led by E. M. BLUESTONE, M.D., New York.

*Thursday, 2:00—Sert Room, Waldorf-Astoria Hotel*

Administrative Panel Round Table Conference (Section A)—a discussion of important questions and problems pertaining to various phases of hospital administration. Conducted by Robert Jolly, Houston.

Administrative Practices: Responsibility for scientific work of hospital. Low cost hospital care versus rising cost of rendering services. Financial security for employees. Selection of hospital administrators. Discussion led by BASIL C. MACLEAN, M.D., Rochester, N. Y.

Hospital Personnel Problems: Personnel turnover. Personnel problems. Hours, pay, and working conditions of personnel. Trends in labor conditions. Discussion led by JOSEPH G. NORBY, Milwaukee, Wis.

Trustees: Maintaining interest of trustees. Responsibility for professional standards. Keeping informed regarding hospital administration. Relations between board of trustees and medical staff. Discussion led by C. MCGREGORY WELLS, JR., Southbridge, Mass.

Business Management: Financial classification of patients. Annual deficit. Reports. Cost per patient day. Discussion led by JAMES R. MAYS, Abington, Pa.

Food Service: Food control. Unit meal costs. Trends in food serving systems. Evaluation of food service. Discussion led by LENNA F. COOPER, New York.

Hospital Auxiliaries: Advantages. Activities. Relationship. Knowledge of hospital. Discussion led by MARY STONE CONKLIN, R.N., Hackensack, N. J.

Linen and Laundry Service: Linen circulation. Unit linen supply. Removal of stains and handling infected linen. Advances in planning and equipping of the hospital laundry. Discussion led by S. FRANK ROACH, Jersey City, N. J.

House Management: Personnel adjustments to meet work load. Disinfection of rooms. Responsibility for cleanliness. Combination housekeeping, dietetics in the small hospital. Discussion led by DORIS L. DUNGAN, Camden, N. J.

The Small Hospital: Personnel. Laboratory and x-ray services. Medical records. Public relations. Discussion led by A. EDWARD A. HUDSON, Waynesboro, Va.

Discussion of questions or problems presented by the assembly.

*Thursday, 2:00—LePerroquet Suite, Waldorf-Astoria Hotel*

Administrative Panel Round Table Conference (Section B)—a discussion of important questions and problems pertaining to various phases of hospital administration. Conducted by R. C. BUERKI, M.D., Madison, Wis.

Professional Practices: Responsibility for professional standards. Major surgery privileges. Medical staff conferences. Criteria for evaluating scientific work of hospital. Discussion led by THOMAS R. PONTON, M.D., Chicago.

Clinical Laboratory: Charges. Routine examinations. Disagreement in diagnosis. Pathological service in the small hospital. Discussion led by PHILIP HILLKOWITZ, M.D., Denver.

Anesthesia: Organization of department. Types of anesthesia. Finances. Legal responsibility. Discussion led by WESLEY BOURNE, M.D., Montreal.

Pharmacy: Minimum standards. Registered pharmacist. Use of proprietary medicine. Economies. Discussion led by M. S. DOOLEY, M.D., Syracuse, N. Y.

Nursing Service: Adjunct nursing personnel. Type of nursing service. Ratio of nurses to patients. Evaluation of Nursing service. Discussion led by GRACE A. WARMAN, R.N., New York.

Medical Social Service: The medical social worker. Minimum standards. Interest of the administration and medical staff. Evaluation of medical social work. Discussion led by MARY H. ROBERTS, Orange, N. J.

Physical Therapy: Essentials of a physical therapy department. Therapeutic physical measures. Fever therapy. Procedure. Discussion led by K. G. HANSSON, M.D., New York.

Occupational Therapy: Advantages. Organization of department. Form of therapy. Financing of department. Discussion led by BYRON M. HARMAN, M.D., Verona, N. J.

Medical Records: Evaluation. Case studies. Recorded pre-operative study. Writing medical records. Discussion led by JESSIE N. HARNED, R.R.L., Durham, N. C.

Discussion of questions or problems presented by the assembly.

# PROGRAMS FOR EVENING SESSIONS

*Presidential Meeting and Convocation—Monday, 8:00 P.M.—Ballroom, Waldorf-Astoria Hotel*

Processional—Officers, Regents, and Honorary Guests.

Invocation. REV. HENRY DARLINGTON, D.D., New York.

Address of Welcome. HENRY W. CAVE, M.D., New York, Chairman, Committee on Arrangements.

Introduction of Foreign Guests. FRANK W. LYNCH, M.D., San Francisco, Vice President.

Address of Retiring President. The American College of Surgeons—Retrospect and Prospect. FREDERIC A. BESLEY, M.D., Waukegan.

Inauguration of Officers:

President: HOWARD C. NAFFZIGER, M.D., San Francisco

First Vice President: VERNON C. DAVID, M.D., Chicago

Second Vice President: FRASER B. GURD, M.D., MONTREAL

Presentation of Initiates for Fellowship. GEORGE CRILE, M.D., Cleveland, Chairman, Board of Regents

Conferring of Fellowships by the President. HOWARD C. NAFFZIGER, M.D., San Francisco.

Conferring of Honorary Fellowships. The President.

Medical Records Honor List and Prize Award. The President.

Annual Oration on Surgery: Our College Mandate—A Tribute to Allen B. Kanavel. WALTER W. CHIPMAN, M.D., Montreal.

*Tuesday, 8:00 P.M.—Ballroom, Waldorf-Astoria Hotel*

Recurrent Hyperthyroidism. RICHARD B. CATTELL, M.D., Boston.

Herniation through the Diaphragm. JOHN J. MORTON, M.D., Rochester, N. Y.

Contractures Due to Burns; Their Prevention and Cure. WILLIAM T. COUGHLIN, M.D., St. Louis.

Results with Repeated Stomach Operations, Especially Gastrojejunal Ulcers. PROF. HANS FINSTERER, Vienna.

*Brooklyn Night—Wednesday, 8:00 P.M.—Ballroom, Waldorf-Astoria Hotel*

Address of Welcome. DONALD E. MCKENNA, M.D., Brooklyn, Chairman, Brooklyn-Long Island Committee on Arrangements, Presiding.

The Radical Operation for Cancer of the Stomach. W. H. OGILVIE, M.D., F.R.C.S.(Eng.), London.

Regional Ileitis:

Surgical Standpoint. CHARLES G. MIXTER, M.D., Boston.

Medical Standpoint. BURRILL B. CROHN, M.D., New York.

Treatment of Bronchiectasis:

Surgical Standpoint. NORMAN S. SHENSTONE, M.D., Toronto.

Medical Standpoint. J. J. SINGER, M.D., Los Angeles.

*Thursday, 8:00 P.M.—Ballrom, Waldorf-Astoria Hotel*

Surgery for Ulcerative Colitis. FRED W. RANKIN, M.D., Lexington, Ky.

The Psychiatrist in Relation to Surgery. FRANKLIN G. EBAUGH, M.D., Denver.

Benign Strictures of the Bile Ducts with a New Method of Treatment. GEORGE E. WILSON, M.B., Toronto.

Fracture Oration: The Evolution of Fracture Treatment. ISIDORE COHN, M.D., New Orleans.

## OPHTHALMOLOGY

*Tuesday and Thursday, 8:00 P.M.—Sert Room, Waldorf-Astoria Hotel*

Graduate Training in Ophthalmology. HARRY S. GRADLE, M.D., Chicago.

A New Visual Phenomenon Useful as a Functional Test in Subjectively Studying Action of Eye Muscles and Retina. CLIFFORD B. WALKER, M.D., Los Angeles.

Present Status of Lacrimal Sac Surgery. RALPH O. RYCHENER, M.D., Memphis.

### OTORHINOLARYNGOLOGY

*Tuesday and Thursaay, 8:oo P.M.—Empire Room, Waldorf-Astoria Hotel*

The Phylogenetic Development of the Ear. JAMES MILTON ROBB, M.D., Detroit.

The Treatment of Otitic Meningitis Due to Streptococcic Infection by Radical Surgery and Sulfanilamide. CARL H. MCCASKEY, M.D., Indianapolis.

Graduate Training in Otolaryngology. ALBERT C. FURSTENBERG, M.D., Ann Arbor, Mich.

Surgical Treatment of Laryngeal Tuberculosis. FLETCHER D. WOODWARD, M.D., HALSTEAD S. HEDGES, M.D., and FRANK B. STAFFORD, M.D., Charlottesville, Va.

A Study of Medical and Surgical Aids to Hearing. JAMES A. BABBITT, M.D., Philadelphia.

## PROGRAMS FOR AFTERNOON SESSIONS

### CANCER SYMPOSIUM

*Tuesday, 2:oo P M.—Ballroom, Waldorf-Astoria Hotel*

Evaluation of Ovarian Sterilization for Breast Cancer. GRANTLEY W. TAYLOR, M.D., Boston. Discussion by RICHARD DRESSER, M.D., Boston.

Surgical Treatment of Laryngeal Cancer. GORDON B. NEW, M.D., Rochester, Minn. Discussion by ALBERT C. FURSTENBERG, M.D., Ann Arbor, Mich.

The X-Ray Treatment of Inoperable Cancer of the Larynx. HENRI COUTARD, M.D., Chicago. Discussion by HAYES E. MARTIN, M.D., New York.

Surgical Treatment of Lung Cancer. ALTON OCHSNER, M.D., New Orleans.

Observations on Palliative Irradiation of Metastatic Tumors in the Lung. ALEXANDER BRUNSCHWIG, M.D., and ANNA HAMANN, M.D., Chicago.

### SYMPOSIUM ON SURGICAL PROCEDURES ON THE HANDICAPPED PATIENT

*Wednesday, 2:oo P.M.—Ballroom, Waldorf-Astoria Hotel*

Factors Determining Selection and Administration of Anesthetics. WESLEY BOURNE, M.D., MONTREAL.

Surgical Problems in Jaundiced Patients. ROBERT S. DINSMORE, M.D., Cleveland.

Surgical Procedures on the Diabetic. LELAND S. MCKITTRICK, M.D., Boston.

Medical Aspects in Pre-operative and Postoperative Care of Diabetic and Cardiac Patients. JAMES E. PAULLIN, M.D., Atlanta, Ga.

### SYMPOSIUM ON INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY

*Thursday, 3:oo P.M.—Ballroom, Waldorf-Astoria Hotel*

Importance of Dusts in Industry and Their Medical Control. LEROY U. GARDNER, M.D., Saranac Lake, N.Y.

Methods of Investigation of Occupational Skin Diseases. LOUIS SCHWARTZ, M.D., New York.

Injuries to the Patella and Surrounding Tissues. WILLIAM R. CUBBINS, M.D., Chicago.

Diagnosis and Therapy of So-called Posttraumatic Neurosis Following Craniocerebral Injuries. DONALD MUNRO, M.D., Boston.

Problems in Rehabilitation after Injury. EDWARD C. HOLMBLAD, M.D., Chicago.

Report of the 1938 Survey. M. N. NEWQUIST, M.D., Chicago.

### SYMPOSIUM ON FRACTURES

*Friday, 2:oo P.M.—Ballroom, Waldorf-Astoria Hotel*

Postgraduate Education in Fractures. GEORGE A. LELAND, JR., M.D., Boston.

The Kinetic Amputation. HENRY H. KESSLER, M.D., Newark.

Double Pin Skeletal Fixation in Fractures of the Leg. R. ARNOLD GRISWOLD, M.D. and GEORGE W. HOLMES, M.D., Louisville.

Fractures of the Bones of the Face. JAMES B. BROWN, M.D., St. Louis.

Conservative Treatment of Fractures. ELDRIDGE L. ELIASON, M.D., Philadelphia.

### SYMPOSIUM ON OBSTETRICS AND GYNECOLOGY

*Friday, 2:00 P.M.—Sert Room, Waldorf-Astoria Hotel*

Certain Aspects of So-called Sterility. ARCHIBALD D. CAMPBELL, M.D., Montreal.

Experience with the Melhado Maneuver for Persistent Posterior Position. GEORGE M. WHITE, M.D., St. John, N. B.

Ovarian Hormones and Carcinogenesis. LUDWIG A. EMGE, M.D., San Francisco.

The Management of Uterine Prolapse by Multiple Plastic Procedures. EDWARD A. SCHUMANN, M.D., Philadelphia.

Wertheim Operation for Cancer of the Uterus. PROF. PAUL WERNER, Vienna.

### SYMPOSIUM ON UROLOGIC INFECTIONS

*Friday, 2:00 P.M.—Le Perroquet Suite, Waldorf-Astoria Hotel*

Obstructive Uropathies. ALEXANDER RANDALL, M.D., Philadelphia.

Problems in Differential Diagnosis between Urologic Lesions and Abdominal Lesions. HERMAN L. KRETSCHMER, M.D., Chicago.

Sympathectomy for the Relief of Vesical Spasm and Pain Resulting from Intractable Bladder Infection. REED M. NESBIT, M.D., Ann Arbor, Mich.

Renal Infections and Nephrolithiasis. GEORGE GILBERT SMITH, M.D., Boston.

Pyelonephritis and Its Treatment. WILLIAM F. BRAASCH, M.D., Rochester, Minn.

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## MIDDAY ROUND TABLE CONFERENCES

*12:00 M. to 1:00 P.M.—Waldorf-Astoria Hotel*

TUESDAY

*Jansen Suite*

Infections in Surgery. MONT ROGERS REID, M.D., Cincinnati, Presiding.

Collaborators: DEAN LEWIS, M.D., Baltimore; URBAN MAES, M.D., New Orleans; MICHAEL L. MASON, M.D., Chicago; ALLEN O. WHIPPLE, M.D., New York.

*Blue Room*

Shock. ALFRED BLALOCK, M.D., Nashville, Presiding.

Collaborators: WILLIAM DEWITT ANDRUS, M.D., New York; NORMAN E. FREEMAN, M.D., Philadelphia; CARL H. LENHART, M.D., Cleveland; DALLAS B. PHEMISTER, M.D., Chicago.

*Carpenter Suite*

The Immediate Repair of Cutaneous Defects. SUMNER L. KOCH, M.D., Chicago, Presiding.

Collaborators: JAMES B. BROWN, M.D., St. Louis; EARL C. PADGETT, M.D., Kansas City, Mo.; GEORGE WARREN PIERCE, M.D., San Francisco; JEROME P. WEBSTER, M.D., New York.

*Assembly Suite*

Thoracic Surgery. WILLIAM F. RIENHOFF, JR., M.D., Baltimore, Presiding.

Collaborators: NORMAN S. SHENSTONE, M.D., Toronto; WALTER ESTELL LEE, M.D., Philadelphia.

WEDNESDAY

*Blue Room*

The Choice of Anesthetic. JOHN S. LUNDY, M.D., Rochester, Minn., Presiding.

Collaborators: WESLEY BOURNE, M.D., Montreal; WILLIS D. GATCH, M.D., Indianapolis; RALPH M. WATERS, M.D., Madison, Wis.

*Jansen Suite*

Craniocerebral Injuries. CLAUDE C. COLEMAN, M.D., Richmond, Presiding.
Collaborators: B. NOLAND CARTER, M.D., Cincinnati; DONALD MUNRO, M.D., Boston.

*Empire Room*

The Surgical Problem of Hypertension. LOYAL DAVIS, M.D., Chicago, Presiding.
Collaborators: IRVINE HEINLY PAGE, M.D., Indianapolis; GEORGE J. HEUER, M.D., New York; REGINALD H. SMITHWICK, M.D., Boston; ALFRED W. ADSON, M.D., Rochester, Minn.

*Assembly Suite*

The Operative Treatment of Hyperparathyroidism. EDWARD D. CHURCHILL, M.D., Boston, Presiding.
Collaborators: J. DELLINGER BARNEY, M.D., Boston; JOHN J. MORTON, M.D., Rochester, N. Y.; DALLAS B. PHEMISTER, M.D.. Chicago.

FRIDAY

*Le Perroquet Suite*

The Treatment of Open Wounds. ROY D. MCCLURE, M.D., Detroit, Presiding.
Collaborators: L. KRAÉER FERGUSON, M.D., Philadelphia; EDWARD L. HOWES, M.D., Washington; MONT ROGERS REID, M.D., Cincinnati.

*Jansen Suite*

The Prevention of Postoperative Pulmonary Complications. EMILE HOLMAN, M.D., San Francisco, Presiding.
Collaborators: CLAUDE S. BECK, M.D., Cleveland; ELLIOTT C. CUTLER, M.D., Boston; WILLIAM F. RIENHOFF, JR., M.D., Baltimore.

*Assembly Suite*

Infections in Obstetrics. JOHN FRASER, M.D., Montreal, Presiding.
Collaborators: FRED L. ADAIR, M.D., Chicago; ALFRED C. BECK, M.D., Brooklyn; GEORGE W. KOSMAK, M.D., New York; ARTHUR H. MORSE, M.D., New Haven.

*Blue Room*

Cancer of the Prostate. HUGH H. YOUNG, M.D., Baltimore, Presiding.
Collaborators: WILLIAM F. BRAASCH, M.D., Rochester, Minn.; ALEXANDER RANDALL, M.D., Philadelphia; GEORGE GILBERT SMITH, M.D., Boston.

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## ASSEMBLY OF INITIATES

*Monday, 3:00 P.M.—Ballroom, Waldorf-Astoria Hotel*

Processional—Initiates, Officers, Regents, and Governors.
Opening Remarks. FREDERIC A. BESLEY, M.D., Waukegan, President
The Program of the American College of Surgeons:
IRVIN ABELL, M.D., Louisville, Vice Chairman, Board of Regents.
BOWMAN C. CROWELL, M.D., Chicago, Associate Director.
MALCOLM T. MACEACHERN, M.D., Chicago, Associate Director.
The Fellowship Pledge. Recital by Initiates.
Greetings to the Initiates. HOWARD C. NAFFZIGER, M.D., San Francisco, President-elect.
Closing Remarks. GEORGE CRILE, M.D., Cleveland, Chairman, Board of Regents.
Signing of the Fellowship Roll by the Initiates.
Reception to Initiates and Fellows.

# PROGRAM FOR THE 1938 CLINICAL CONGRESS

ARRANGED IN THE FOLLOWING SUBDIVISIONS: GENERAL SURGERY, OBSTETRICS AND GYNECOLOGY, SURGERY OF BONES AND JOINTS, GENITO-URINARY SURGERY, FRACTURES AND TRAUMATIC SURGERY, THORACIC SURGERY, NEUROSURGERY, PLASTIC AND FACIOMAXILLARY SURGERY, OPHTHALMOLOGY, OTOLARYNGOLOGY.

## NEW YORK—GENERAL SURGERY

### *Monday*

#### BELLEVUE HOSPITAL

E. A. ROVENSTINE and staff—2. Symposium on anesthesia: Anesthetic management of patients with hyperactive carotid sinus reflexes; therapeutic nerve blocks, for angina, intractable pain, etc.; demonstration of the technique of oropharyngeal insufflation of oxygen.

#### BETH ISRAEL HOSPITAL

HARRY E. ISAACS and staff—2. Operations, with particular reference to diseases of the gall bladder. Dry clinic: Cholecystectomy without drainage; common duct obstruction; resectable liver tumors.

#### FLOWER-FIFTH AVENUE HOSPITAL

J. H. FOBES and associates—2. Tumor clinic.
THOMAS H. MCGAVACK. Lymphosarcoma. Medical aspects of suprarenal tumors.
J. C. HOWARD. Hodgkins disease.
F. J. BORELLI and W. E. YOULAND.
L. R. KAUFMAN. Surgical aspects of suprarenal tumors.
L. C. REID and F. D. SPEER. Pathology.
G. H. ADLER. Endocrine aspects.
J. H. FOBES. Report of angiosarcoma of the cauda equina; intensive x-ray treatment and operation; result at end of 21 years, with presentation of patient.
J. H. FOBES, W. E. YOULAND, and J. C. HOWARD. Symposium on breast cancer.
J. H. FOBES, D. B. HILL, J. C. HOWARD, and W. E. YOULAND. Fibrolipoma of the cecum with intussusception; fibrolipoma of the rectum.

#### FORDHAM HOSPITAL

E. R. CUNNIFFE, R. E. WALSH, and ALFRED G. FORMAN—2. Operative and dry clinics.

#### GOUVERNEUR HOSPITAL

R. F. CARTER and R. B. LOBBAN—2. Diagnosis and surgical management of gall bladder disease.

#### HARLEM HOSPITAL

LOUIS T. WRIGHT—2. Operations and ward rounds.
CLARENCE P. HOWLEY—2. Operations and ward rounds.

#### LENOX HILL HOSPITAL

CARL EGGERS, OTTO C. PICKHARDT, DE WITT STETTEN, and staffs—2. Operations.
Staff—2. Symposium on gastric and duodenal ulcer and associated lesions.
WILLIAM H. STEWART. Roentgen diagnosis of peptic ulcer by the modern mucosal method.
MAX EINHORN. Intubation treatment of peptic ulcer.
ABRAHAM L. GARBAT. Ambulatory treatment of peptic ulcer.
HENRY A. RAFSKY. Medical treatment of pyloric obstruction.
CARL EGGERS. Gastro-enterostomy in peptic ulcer.
DEWITT STETTEN. End results after resection for peptic ulcer.
OTTO C. PICKHARDT. Treatment of associated lesions.
HERMANN FISCHER—2. Exhibition of moulages of pathological specimens of gastro-intestinal tract.

#### METROPOLITAN HOSPITAL

S. T. GLASSER and ALBERT LESSER—1:30. Injection treatment of varicose veins.

#### MISERICORDIA HOSPITAL

ARTHUR S. MCQUILLAN—2. Symposium on thyroid gland surgery; study of 2,000 cases.
WILLIAM T. DORAN, SR.—3:30. Surgical judgment in procedures of the upper abdomen.

#### MOUNT SINAI HOSPITAL

RALPH COLP, PERCY KLINGENSTEIN, SIGMUND MAGE, and JOSEPH STENBUCK—1:15. Operations. Dry clinic: Pancreatic reflux, palliative subtotal gastrectomy for juxtacardial gastric ulcer, study of failures after gastroenterostomy.

#### NEW YORK CITY HOSPITAL

LYMAN W. CROSSMAN and JAMES H. KIDDER—2. Operations.

#### NEW YORK FOUNDLING HOSPITAL

GEORGE R. STUART and staff—2. Unusual surgical cases. roentgenological, pathological and surgical aspects.

#### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

EDWARD W. PETERSON—2. Operations.

#### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

FRANK C. YEOMANS—1:30. Proctological operation.

#### ST. LUKE'S HOSPITAL

Staff—2:30. Symposium on thyroid diseases.
C. L. GILBERT. Radiotherapy in toxic goiter.
E. HERBERT, JR. Incidence of malignancy in solitary adenoma of thyroid.
MORRIS K. SMITH. Amount of remnant in operations for diffuse toxic goiter.
G. M. GOODWIN. Discussion of postoperative tetany.

#### Scientific Exhibitions—Daily

Tumor exhibit. F. C. WOOD, Director Department of Pathology.
New growths of the respiratory and gastro-intestinal tracts. E. J. RYAN, Director Department of X-Ray Diagnosis.

### ST. VINCENT'S HOSPITAL

MAURICE C. O'SHEA—2. Severed tendons and nerves of the hand and forearm.
ANTHONY ROTTINO—2. Pathological demonstration.

### SYDENHAM HOSPITAL

MILTON BODENHEIMER—2. Operations: Various types of thyroid disease.

## *Tuesday*

### BABIES HOSPITAL

EDWARD J. DONOVAN, WILLIAM G. HEEKS, LOUIS M. ROUSSELOT, and GEORGE H. HUMPHREYS—9. Operations.

#### Dry Clinics

EDWARD J. DONOVAN. Congenital duodenal obstructions.
WILLIAM G. HEEKS. Treatment of acute empyema.
LOUIS M. ROUSSELOT. Treatment of general peritonitis in children.
GEORGE H. HUMPHREYS. Retroperitoneal infections in children.

### BELLEVUE HOSPITAL

ARTHUR BURDICK and staff—9:30. Operations.
J. A. MCCREERY and staff—9:30. Operations.
GUILFORD DUDLEY and staff—9:30. Operations.
Staff—2. Symposium on surgical diseases of the stomach.
JACOB BUCKSTEIN. Roentgenologic diagnosis of gastric, duodenal and gastric jejunal ulcers.
JOHN A. MCCREERY. Operative management of secondary gastric and duodenal ulceration including marginal ulcers.
GUILFORD S. DUDLEY. Inflammatory and benign tumors of the stomach and their surgical management.
ARTHUR M. WRIGHT, FRANCES H. BOGATKO, and WILLIAM H. BARBER. Failures after gastrojejunostomy; clinical and experimental studies.
ROLAND L. MEIER. Treatment of gastric ulcer.
REYNOLD E. CHURCH. Surgical treatment of massive hemorrhage.
J. WILLIAM HINTON. Surgical treatment of chronic duodenal ulcers.

### FLOWER-FIFTH AVENUE HOSPITAL

HERBERT C. CHASE—9. Breast operations and motion pictures.
H. D. FURNISS and W. P. ECKES—9. Cases of intestinal obstructions.
EARL H. EATON and LOUIS PALERMO—9. Treatment of burns with horse serum and tannic acid.
W. G. CRUMP—9. Carcinoma of rectum.
J. H. FOBES—9. Carcinoma of colon.
JOSEPH A. SILEO—9. Thrombosis and embolism, postoperative care.
L. R. KAUFMAN and J. H. FOBES and staffs—2. Operations.

### FORDHAM HOSPITAL

E. R. CUNNIFFE, R. E. WALSH, and ALFRED G. FORMAN—2. Operative and dry clinics.

### GOUVERNEUR HOSPITAL

FRANCIS M. CONWAY—9. Diverticulitis of the colon.
MORRIS M. BERCK—9. Diagnosis and treatment of penetrating wounds of the esophagus.
JOSEPH GIRSDANSKY—2. Injection treatment of hernia and varicose veins.

### HARLEM HOSPITAL

JOSEPH B. STENBUCK—9. Operations and ward rounds.
LEON GINZBURG—9. Operations.
CHARLES S. B. CASSASA, JOSEPH B. STENBUCK, FARROW R. ALLEN, IRA FINK, ROBERT E. CARTER, NORMAN F. LASKEY, WILLIAM H. MENCHER, BENJAMIN N. BERG, FRANCIS X. TIMONEY, JOHN R. BRUCKNER, ROBERT S. WILKINSON, and WILLIAM SNOW—10:30. Symposium on subcutaneous and penetrating wounds of the thorax and abdomen.

### HOSPITAL FOR JOINT DISEASES

MILTON BODENHEIMER and staff—9. Operations: Thyroid surgery.
ABRAHAM J. BELLER and staff—9. Operations: Gastrectomy for carcinoma of the stomach; abdominoperineal resection for carcinoma of the rectum.
HARRY GOLDMAN and staff—9. Proctological operations.

### KNICKERBOCKER HOSPITAL

GEORGE H. SEMKEN—9. The cancer problem. Symposium with lantern slides.

### LENOX HILL HOSPITAL

Staff—9. Dry clinic: Affections of the thyroid gland and complications.
ARTHUR F. KRAETZER. General survey of thyroid disease.
JACOB GEIGER. Value of basal metabolism tests in thyroid disorders.
A. S. BLUMGARTEN. Medical treatment of thyroid disorders and associated endocrine disturbances, including diabetes.
THOMAS K. DAVIS. The psychiatric manifestations in hyperthyroidism.
CLARENCE DE LA CHAPELLE. Cardiovascular problems in the thyroid diseases.
PAUL K. SAUER. The thyroid in relation to heart disease.
WALTER T. STENSON. Postoperative care of patients with hyperthyroidism.
ERICH FRICKE. Postoperative results in cases with hyperthyroidism.
FRANCIS M. DONEHUE. Postoperative tetany and its treatment.
OTTO C. PICKHARDT. The use of A.T. 10 in postoperative tetany.
CARL EGGERS—2. Esophageal lesions with demonstration of patients.
WM. H. STEWART and staff—2. Cinefluorographic demonstration of esophageal lesions.
HERBERT W. MEYER—2. Reconstruction operations for epithelioma of the face.
ARTHUR STEIN—2. Lesions of the vulva; lantern slides.

### LINCOLN HOSPITAL

KIRBY DWIGHT—9. Operations on the colon. Dry clinic: Peritonitis and ileus.
FREDERICK H. AMENDOLA, SAMUEL EPSTEIN, and REUBEN GILBERT—11:15. Diseases of the thyroid.

### LUTHERAN HOSPITAL

ALFRED G. FORMAN and staff—9. Operations.
Staff—2. Dry clinic.
JOHN P. BRUCKNER. Ludwig's angina.
CHARLES S. CASSASA. Intestinal obstruction.
ANGELO A. ZINGARO. Epiphyseal fracture.
LOUIS PERROTTA. Technique of spinal anesthesia and end results.

### MEMORIAL HOSPITAL

BRADLEY L. COLEY—9. Amputation of leg for osteogenic sarcoma.
FRANK E. ADAIR—10. Radical amputation of breast for carcinoma.

## METROPOLITAN HOSPITAL

J. H. Fobes and L. R. Kaufman, and staffs—9. Symposium: Surgery of the stomach, liver, spleen and pancreas.
T. B. Weinberg. Roentgenological aspects.
Dr. McNeill. Gastroscopy.
Linn J. Boyd and Thomas H. McGavack. Medical aspects.
J. H. Fobes and L. R. Kaufman. Operations.
J. H. Fobes and John S. O. Herrlin, Jr. Demonstration of cases of pancreatic cysts.
Roy Upham, D. B. Hill and staffs. Medical aspects of gastric ulcer.
Staff—1:30. Symposium on gastro-intestinal surgery.
D. B. Hill, J. H. Fobes, and J. C. Howard. Lesions of the lesser curvature of the stomach, medical aspect, surgical aspect, motion pictures and lantern slides.
Roy Upham, J. C. Howard, A. Saccone, and L. R. Kaufman. General management of gastric and duodenal ulcer, medical aspect, roentgenological and pathological aspects, surgical treatment.
Charles A. Halberstam, Edward J. McCabe, and W. W. Johnson. Report on perforation of gastric and duodenal ulcers.
L. R. Kaufman, J. H. Fobes, John S. O. Herrlin, Jr., William P. Eckes, and Edward J. McCabe. The jaundice problem, report by the complete surgical staff.
J. H. Fobes and staff. Report of two cases of solitary liver abscess.
H. D. Furniss and W. P. Eckes. Intestinal obstruction.
John S. O. Herrlin, Jr. Discussion of the water balance in treatment.

## MISERICORDIA HOSPITAL

Saul A. Ritter—10:30. Tuberculosis of ileocecal valve simulating malignancy, and other unusual surgical cases.
Lester Breidenbach—2. Phlebectomy for thrombosis.
Maurice J. Costello—3. Surgical neoplasms of skin.
Peter Rizzo—3:30. Devices in skeletal surgery.

## MONTEFIORE HOSPITAL

A. A. Berg and staff—2. Operations on the colon.

## MORRISANIA CITY HOSPITAL

J. Lewis Amster—9. Operation demonstrating regional anesthesia.

## NEW YORK CITY CANCER INSTITUTE

Ira I. Kaplan and associates—2. Symposium on cancer.
Ira I. Kaplan. Treatment of carcinoma of the cervix in advanced cases.
Braham H. Golden. Palliative mastectomy.
George A. Cashman. Genito-urinary malignancy, especially carcinoma of the penis.
Rudolph V. Gorsch. Colostomy.
Angelo M. Sala. Pathology of advanced malignancy.
David E. Ehrlich. Pendant mastography in the diagnosis of carcinoma of the breast.

## NEW YORK CITY HOSPITAL

Frederic Bancroft and Margaret Stanley-Brown—9. Thrombosis; thrombophlebitis; embolism.

## NEW YORK HOSPITAL

George J. Heuer and staff—9. Operative and dry clinics.
George J. Heuer. Surgical treatment of hypertension.
William DeW. Andrus. Splenectomy.
William F. MacFee. Surgical lesions of the mouth and jaws.
Ralph F. Bowers. Results in 900 thyroidectomies.
Bronson S. Ray. Organization of a follow-up department.
N. Chandler Foot. Contribution of a laboratory of surgical pathology to a surgical service.
Frank Glenn. Surgery of common bile duct.

## NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

Anna Hubert, Mary L. Edwards, and Frances H. Bogatko—9. Surgical follow-up clinic.
Elise S. L'Esperance—2. Tumor conference.

## NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

Carl Eggers—9. Breast clinic (Skin and Cancer unit).
Thomas H. Russell—2. Operations.

## NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

Frederick C. Keller—10. Cadaver demonstration, surgical anatomy.
Robert E. Brennan—11. Operations.
Jerome M. Lynch—1:30. Operations, proctological.
Richard Kovacs—2:30. Lecture on physical therapy.
Edward L. Kellogg—3:30. Operations.

## PRESBYTERIAN HOSPITAL

Allen O. Whipple and Louis M. Rousselot—9. Splenectomy.
William Barclay Parsons and Lawrence W. Sloan—9. Thyroid operations.
Walter W. Palmer, William Barclay Parsons, Lawrence W. Sloan, Bertram J. Sanger, and Arthur P. Stout—10. Symposium on thyroid disease.
Allen O. Whipple, William P. Thompson, Kenneth R. McAlpin, R. West, Louis M. Rousselot, and Robert H. E. Elliott—2. Symposium on blood dyscrasias and splenopathies.

## ROOSEVELT HOSPITAL

Staff—9. Symposium on surgical management of intractable ulcerative colitis.
Thomas T. Mackie. Medical aspects.
Henry W. Cave. Operation, with discussion.
Lawrence Sophian. Pathological aspects.
Staff—2. Symposium on ulcers and carcinoma of the stomach.
Howard F. Shattuck. Medical aspects.
William H. Boone. Roentgen diagnosis.
Condict W. Cutler, Jr. Indications for surgical treatment.
Gordon P. McNeer. Demonstration of gastroscopy.
Grant P. Pennoyer and Julian M. Freston—3:30. Demonstration of various types of peripheral vascular diseases with differential diagnosis and the results of treatment.

## ST. FRANCIS' HOSPITAL

Alexander Nicoll and staff—9. Cholecystectomy; cholecystectomy with exploration of common duct.
Charles Vejvoda and staff—9. Cholecystectomy; appendectomy; herniorrhaphy.

## ST. LUKE'S HOSPITAL

Henry H. M. Lyle, John Douglas, Edward J. Donovan, William F. MacFee, Morris K. Smith, and staff—9. Operative and dry clinics.
B. R. Shore. Surgical treatment of epitheliomas of the face.

WILLIAM F. MACFEE. Obstruction of the small intestines.
E. D. TRUESDELL. The prognosis in silent gallstones.
EDWARD J. DONOVAN. Congenital diaphragmatic hernia.
WILLIAM G. HEEKS and WILLIAM T. GIBB, JR. The value of gastroscopy in diagnosis; demonstration of gastroscopy.

### ST. VINCENT'S HOSPITAL

JOSEPH A. BRADY, GEORGE R. STUART, HENRY V. WALSH, FRANK J. MCGOWAN, BERNARD D. HANNAN, and FRANCIS X. TIMONEY—9. Operations.
RAYMOND P. SULLIVAN, CONSTANTINE J. MACGUIRE, JR., LOUIS F. SANMAN, CLARENCE P. HOWLEY, and JOHN H. MORRIS—9. Operations.

### SYDENHAM HOSPITAL

MILTON BODENHEIMER—3. Dry clinic: Stab and bullet wounds of the chest and abdomen.

### UNITED STATES MARINE HOSPITAL

L. A. PALMER and staff—10:30. Symposium on gastric surgery.
L. A. PALMER and staff. Surgical aspects.
C. R. SMITH. Pathological aspects.
F. LIBERSON. X-ray reports.
J. A. BRASFIELD and HAROLD KELMAN—10:30. Symposium on back injuries: Surgical aspects; neuropsychiatric aspects.
HAROLD KELMAN and S. P. COOPER—2. Symposium on peripheral vascular disease.
L. A. PALMER and staff—2. Operative and dry clinic: Hernia.

## *Thursday*

### BEEKMAN STREET HOSPITAL

Staff—9. Symposium on abdominal emergencies from various clinical viewpoints; ward rounds.
ARTHUR H. TERRY, JR. The acute abdomen in the diabetic.
ROBERT H. KENNEDY. Ovarian conditions contributing to an acute abdomen.
SIGMUND MAGE. Retroperitoneal injury simulating acute abdomen.
JAMES H. HEYL. Pseudopancreatic cyst following abdominal trauma.
ELIAS RUBIN. Genito-urinary tract injuries.
CHARLES J. OPPENHEIM. Coronary thrombosis.
THOMAS M. LOWRY. Acute allergic abdomen.
MYRON A. SALLICK. Perforated peptic ulcer.

### BELLEVUE HOSPITAL

Staff—9:30. Symposium on surgery of the thyroid.
RODERICK V. GRACE and CARNES WEEKS. Total thyroidectomy for heart disease, four-year results; progressive exophthalmus following Graves' disease; aberrant thyroid, two cases; interval surgery in the patient with hyperthyroidism.
RUSSEL H. PATTERSON. Prevention and treatment of complications in goiter surgery.
H. M. WERTHEIM. Demonstration of cervical plexus anesthesia block, lantern slides.
H. A. D. O'CONNOR and JOSEPH NASH. Evaluation of postoperative thyroid results.
ARTHUR S. MCQUILLAN. Ossification of a thyroid adenoma; Huerthle-cell tumor of the thyroid gland; Hashimoto's disease of the thyroid gland; epithelioid carcinoma of the thyroid gland; hyperparathyroidism; postoperative tetany, treatment.
ARTHUR M. WRIGHT and staff—9:30. Operations.
FENWICK BEEKMAN and staff—2. Symposium on children's surgery: Treatment of fractures, congenital anomia, burns, problem cases.
IRA I. KAPLAN and associates—2. Operative and dry clinics: Irradiation treatment of malignant and benign lesions.
IRA I. KAPLAN. Treatment of carcinoma of the cervix with radium.
RIEVA ROSH. Surface lesions including carcinoma of the lip.
DOROTHY BELL. Primary and postoperative carcinoma of the breast.
MILTON FRIEDMAN. Tumors of the upper respiratory tract.
SIDNEY RUBENFELD. Lymphoblastomas.

### FLOWER-FIFTH AVENUE HOSPITAL

L. R. KAUFMAN and J. H. FOBES, and staffs—2. Operations.
J. H. FOBES and associates—2. Symposium on gastrointestinal surgery; lesions of the lesser curvature of the stomach.
D. B. HILL. Medical consideration.
J. H. FOBES and J. C. HOWARD. Surgical consideration; motion pictures and lantern slides; general management of gastric and duodenal ulcer.
ROY UPHAM. Medical consideration.
J. C. HOWARD. Roentgenology.
L. C. REID. Pathology.
L. R. KAUFMAN. Surgery.
CHARLES A. HALBERSTAM, EDWARD J. MCCABE, and WILLIAM WARNER JOHNSON. Report of perforation of gastric and duodenal ulcers.
J. H. FOBES, L. R. KAUFMAN, JOHN S. O. HERRLIN, JR., W. P. ECKES, and EDWARD J. MCCABE. The jaundice problem.
H. D. FURNISS and W. P. ECKES. Intestinal obstructions.
JOHN S. O. HERRLIN, JR. Discussion of water balance.

### FORDHAM HOSPITAL

ALEXANDER NICOLL, JAMES H. KENYON, and LOUIS MARTON—9 and 2. Operative and dry clinics.

### GOUVERNEUR HOSPITAL

J. F. ERDMANN—9. Operations.

### HARLEM HOSPITAL

RALPH H. YOUNG—9. Operations.
CHARLES S. B. CASSASA—9. Operations.
LOUIS T. WRIGHT, JOSEPH G. LEVY, SOLOMON WEINTRAUB, FREDERICK A. KASSEBOHM—10. Symposium on lymphopathia venereum.

### LENOX HILL HOSPITAL

OTTO C. PICKHARDT, CARL EGGERS, DEWITT STETTEN, and staffs—9. Operations.

### LINCOLN HOSPITAL

EDWARD D. TRUESDELL—9. Operations on biliary tract.
CHARLES S. ROGERS—10:30. Dry clinic: Children's surgery.
JAMES R. LINCOLN, JACOB FRIEDMAN, and LEONARD ORENS—11:15. Surgical complications of diabetes.

### MEMORIAL HOSPITAL

GEORGE E. BINKLEY—9. Resection for carcinoma of rectum.

GEORGE T. PACK—9. Resection for gastric carcinoma.
Staff—11. Cancer conference.

### METROPOLITAN HOSPITAL

THOMAS H. MCGAVACK, CHARLES A. HALBERSTAM, S. T. GLASSER, and ALBERT LESSER—9. Peripheral vascular diseases, with demonstration of arteriography, oscillometry, Landis test and histamine test.
DAVID WEEKS and associates—9. Report on experimental work in the production of renal hypertension and operative procedures for relief.
J. H. FOBES, L. R. KAUFMAN, and staffs—9. Operations.

### MOUNT SINAI HOSPITAL

JOHN H. GARLOCK, LEON GINZBURG, WILLIAM H. MENCHER, and MOSES SWICK—1:15. Operations: Colonic surgery; obstructive jaundice. Dry clinic: Ulcerative colitis; carcinoma of the esophagus.

### NEW YORK CITY HOSPITAL

PAUL K. SAUER and staff—9. Operative and dry clinics.
LESTER BLUM—9. Experimental cardiac surgery.
W. G. TERWILLIGER—9. Varicose veins.
ROBERT T. FINDLAY—9. Surgery in the poor-risk aged.

### NEW YORK HOSPITAL

GEORGE J. HEUER and staff—9. Operative and dry clinics.
FRANK GLENN. Acute cholecystitis.
WILLIAM F. MACFEE. Carcinoma of the large bowel.
RALPH F. BOWERS. Terminal ileitis.
CRANSTON W. HOLMAN. Pre- and postoperative studies of gastric secretion.
WILLIAM M. COOPER. Carcinoma of the ampulla of Vater.
J. HERBERT CONWAY. Leiomyosarcoma of the stomach.

### NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

ANNA HUBERT, MARY A. JENNINGS, and ISABEL KNOWLTON—9. Operations.
FRANCES BOGATKO—9. Varicose vein clinic.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

CARL EGGERS—9. Dry clinic: Surgery of the head and neck.
CHARLES G. HEYD—9. Operations.
EDWARD W. PETERSON—9. Operations.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

JOHN J. MCGRATH—11. Operations.
VINCENT HURLEY—1:30. Proctology, cadaver demonstration.
J. E. HAMMETT—2:30. Operations.

### PRESBYTERIAN HOSPITAL

BEVERLY C. SMITH, DAVID C. BULL, LOUIS BAUMAN, and BYRON STOOKEY—9. Operations. Symposium on vascular disturbances of the extremities.
JOHN M. HANFORD, ARTHUR P. STOUT, CUSHMAN D. HAAGENSEN, MAURICE LENZ, and THEODORE P. EBERHARD—2. Symposium on therapy of tumors of head and neck.

### ROOSEVELT HOSPITAL

HOWARD A. PATTERSON—9. Thyroid clinic: Followed by demonstration of cases with discussion of recurrent and persistent hyperthyroidism.

### HOSPITAL FOR RUPTURED AND CRIPPLED

CARL G. BURDICK and associates—9. Symposium on hernia.
JOHN E. SULLIVAN. Postoperative complications.
RODERICK V. GRACE and VANSEL S. JOHNSON. Results of herniotomy in patients over fifty years of age.
FENWICK BEEKMAN. Undescended testicle.
NORMAN L. HIGINBOTHAM. Division of cord.
BRADLEY L. COLEY. Injection treatment.
DAVID GILLESPIE. Fascial sutures in the repair of hernia.
ROLAND L. MAIER. Silk sutures in the repair of hernia.
WILLIAM CRAWFORD WHITE—9. Demonstrations of cases of carcinoma of the thyroid gland with discussion of treatment.
LEWIS S. BOOTH, MALCOLM H. MUNKITTRICK, and PAUL M. WOOD—2. Demonstration of modern trends in anesthesia.

### ST. FRANCIS' HOSPITAL

ROBERT B. LOBBAN and staff—9. Subtotal gastrectomy; stomach cases.

### ST. LUKE'S HOSPITAL

HENRY H. M. LYLE, JOHN DOUGLAS, EDWARD J. DONOVAN, WILLIAM F. MACFEE, MORRIS K. SMITH, and staff—9. Operations.

### ST. VINCENT'S HOSPITAL

EDWARD V. DENNEEN, FRANK J. SERAFIN, WILLIAM P. MACNAMARA, HENRY A. WAHN, and JAMES F. BROWN—9. Operations.
RAYMOND P. SULLIVAN, JOHN A. LAWLER, MAURICE C. O'SHEA, THOMAS C. CASE, FRANCIS M. CONWAY, and JOHN A. FALLON—9. Operations.
RAYMOND P. SULLIVAN—2. Malignant and non-malignant lesions of colon, surgical aspects.
WILLIAM W. MAVER. X-ray demonstrations.
ANTHONY ROTTINO. Pathological demonstration.
Staff—2:45. Symposium on hernia.
CLARENCE P. HOWLEY. Diaphragmatic hernia.
JOHN A. LAWLER. Paraduodenal and paravesical hernias.
Staff—3:30. Symposium on perforated ulcer.
FRANK J. MCGOWAN. Operative procedure.
EDWARD V. DENNEEN. Five-year operative experience.
J. RAYMOND LUTZ. Medical management.
JOHN H. MORRIS. Follow-up experience.

### SYDENHAM HOSPITAL

CULLEN ADLERBLUM—9. Dry clinic: Varicose vein injections.
MEYER H. FREUND—10:30. Operations: Perineal excision of the rectum for carcinoma; anorectal operations.
LESTER J. UNGER—2:30. Operations: Blood transfusion—Unger method.

### UNITED STATES MARINE HOSPITAL

Staff—10:30. Symposium on appendicitis.
L. A. PALMER and staff. Surgical aspects.
C. R. SMITH. Pathological aspects.
J. W. KENNEDY. X-ray findings.
C. FERGUSON and R. MEE—10:30. Symposium on rectal surgery.
S. P. COOPER—10:30. Hernia operations.

### VETERANS ADMINISTRATION HOSPITAL

FREDERICK W. BANCROFT—9. Operations: Gastric resection for pyloric obstruction, 2 cases. Discussion of thrombosis and embolism.

ALLEN G. FULLER—9. Operations: Carcinoma of the rectum, second stage.
H. J. BALLEN, E. P. HALL, and J. P. DELANEY—10. Operations: Inguinal hernia repair, with demonstration of sliding bladder, 2 cases; end result cases, stomach and intestinal, nonmalignant.
DEFORREST BALLOU, JR.—10. Operations: Laryngofissure, partial laryngectomy. Dry clinic: Carcinoma of the larynx.
B. F. HAYDEN, Manager; CARLETON BATES, Clinical Director; JAMES EWING, FRANK E. ADAIR, and FRED W. STEWART, Consultants—2. Tumor conference.
ALLEN G. FULLER. Carcinoma of the stomach.
E. LEVY. Carcinoma of the lung.
J. P. PALMER. Carcinoma of lip and tongue.
W. G. CHRISTOFFERSEN. Basal cell carcinoma and carcinoma of the rectum.
CHARLES F. BLOOM. Collaborated material.
R. C. HENDERSON. Collaborated material.
DEFORREST BALLOU, JR. Carcinoma of the larynx.
H. HERSCHER. Hodgkins disease and lymphosarcoma.

*Friday*

BELLEVUE HOSPITAL

Staff—9:30. Symposium on gall bladder surgery.
CONSTANTINE J. MCGUIRE. Treatment of acute cholecystitis.
FRANK COTUI. Intravenous medication in gall bladder disease.
IRA I. KAPLAN. Radiation therapy in disease of the biliary tract.
WILLIAM T. DORAN. Management of the gall bladder patient.
ERNEST W. LAMPE. Acute pancreatitis in relation to biliary tract disease.
JOHN E. SUTTON, JR. Jaundice and common duct stone.

BETH ISRAEL HOSPITAL

PERCY KLINGENSTEIN and staff—2. Operations: Graves' disease; gastroduodenal ulcer. Dry clinic: Pre-operative radiation in carcinoma of the breast.
I. KROSS—2. Dry clinic: Obstructive lesions of the small intestine. Case presentations.
SAMUEL MUFSON—2. Dry clinic: Unusual pre-operative complications of hernia.

WILLIAM BOOTH MEMORIAL HOSPITAL

WILLIAM T. KENNEDY—9. Surgical treatment of incontinence of urine in women.
JOHN ROGERS and ARTHUR MCQUILLAN—10. Thyroid clinic.
GEORGE W. KOSMAK and N. GILBERT SEYMOUR. Demonstration of medical and surgical facilities in a small private hospital run by the Salvation Army for people of moderate means.

FLOWER-FIFTH AVENUE HOSPITAL

J. CLIFFORD HAYNER, DEW. R. S. BARNES, and W. L. PRIMACOVE—9. The use of lemon oil as a skin antiseptic.
JOHN S. O. HERRLIN, JR.—9. Infections of the hand; wax models; motion pictures.
J. H. FOBES and L. R. KAUFMAN and staffs—2. Operations.
J. H. FOBES and S. T. GLASSER—2. The use of ox fascia in hernia operations.
J. C. HAYNER—2. Demonstration of new method in hernia operations.
L. R. KAUFMAN and W. W. JOHNSON—2. Demonstration of wire sutures.
W. W. JOHNSON and ALBERT LESSER—2. Experimental work on dogs.
J. D. NORRIS—2. Studies in wound healing—dehiscence.
DAVID WEEKS and associates—2. Symposium on hypertension. Report on experimental work in the production of the renal type and operative procedures for relief.
WILLIAM H. BISHOP and J. H. FOBES—2. Intussusception of appendix, outside of anus, removal; demonstration with lantern slides.
J. H. FOBES, CHARLES HALBERSTAM, and staff—2. The present status of appendicitis, demonstration of a new operation; motion pictures.
DONALD E. BRACE and staff—2. Contribution of a department of anesthesia to a surgical service.
L. C. REID—2. Contribution of a laboratory of surgical pathology to a surgical service.

FRENCH HOSPITAL

ARTHUR M. WRIGHT and staff—9. Symposium on diseases of the gall bladder. Cholecystitis in elderly people treated by cholecystgastrostomy; stone in the common duct, poor operative risk; cholecystogastrostomy, patient living ten years later; choledochoduodenostomy for recurrent obstruction of common bile duct, rubber tube implantation, five months; resection of sigmoid for carcinoma complicated by intestinal obstruction by gall stones; pathological discussion by Dominic Anthony DeSanto.

FORDHAM HOSPITAL

E. R. CUNNIFFE, R. E. WALSH, and ALFRED G. FORMAN—2. Operative and dry clinics.

GOUVERNEUR HOSPITAL

MIGUEL G. ELIAS—2. Perforated gastric and duodenal ulcer.
F. J. MCGOWAN—2. Discussion of above.
BORRIS KORNBLITH—2. Lymphogranuloma venereum.

HARLEM HOSPITAL

Staff—9. Dry clinics.
CHARLES S. B. CASSASA, ALEXANDER ALTSCHUL and staff. Thyroid disease.
LEON GINZBURG. Intestinal obstruction.
JAMES C. WHITAKER. Fat embolism.
AUBRE DEL. MAYNARD. Treatment of burns.
U. CONRAD VINCENT. Surgical lesions of the terminal ileum, cecum, and the ascending colon.

KNICKERBOCKER HOSPITAL

JOHN V. BOHRER, C. JOSEPH DELANEY and PRO. V. PREWITT—9. Operative and dry clinic: Surgery of the stomach and gall bladder.

LENOX HILL HOSPITAL

Staff—10:30. Symposium on lesions of the colon.
WILLIAM H. STEWART. Differential diagnosis between carcinoma, diverticulitis and sigmoiditis.
CARL EGGERS. Diverticulitis and sigmoiditis.
DEWITT STETTEN. Carcinoma of the colon and its radical treatment.
HERBERT W. MEYER. Radical operations for carcinoma of the rectum.
JOHN C. A. GERSTER. Conservative operations for carcinoma of the rectum.
Exhibition of moulages of pathological specimens of the gastro-intestinal tract.

LINCOLN HOSPITAL

BRADLEY L. COLEY—9. Management of bone tumors in a general hospital.

BENJAMIN SHERWIN—9:45. Complications in hernia operations.
PETER VOGEL—10:45. Management of a blood bank in a municipal hospital.

### MEMORIAL HOSPITAL

HAYES E. MARTIN—9. Hemilaryngectomy for carcinoma.
GEORGE T. PACK—9. Intrascapulothoracic amputation for melanoma.
Staff—10. Demonstration of x-ray and radium equipment. Inspection of new Memorial Hospital.

### METROPOLITAN HOSPITAL

J. H. FOBES and L. R. KAUFMAN, and staffs—9. Operations.
Staff—9. Dry clinics.
H. D. FURNISS and W. P. ECKES. Discussion of intestinal obstruction.
JOHN S. O. HERRLIN, JR. Water balance.
J. H. FOBES and DR. CHASE. Breast.
J. H. FOBES. Discussion of ox fascia in the repair of hernias.
L. R. KAUFMAN, W. W. JOHNSON, and ALBERT LESSER. Demonstrations of wire sutures in hernia with a report of experimental work on dogs.
J. H. FOBES, J. CLIFFORD HAYNER, and CHARLES A. HALBERSTAM. Report on the present status of appendicitis.
J. WALSH. Report on the value of rebound tenderness.

### MORRISANIA CITY HOSPITAL

GEORGE E. MILANI—2. Operation for repair of large hernias by fascial flaps.
WILLIAM KLEIN—3:30. Treatment of acute mesenteric lymphadenitis.

### NEW YORK CITY CANCER INSTITUTE (Welfare Island Hospital)

IRA I. KAPLAN and associates—2. Symposium on cancer.
IRA I. KAPLAN. Demonstration of cancer cases.
LIONEL S. AUSTER. Surgical problems of advanced cancer, illustrated by specific cases.
DAVID E. EHRLICH. Interesting roentgenograms of advanced cancer cases.

### NEW YORK CITY HOSPITAL

ISIDORE KROSS and staff—9. Operative and dry clinics.

### NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

MARY L. EDWARDS, EMMA S. ARONSON, and MARGARET STANLEY-BROWN—9. Operations.
ASTA J. WITTNER and SOPHIE SPITZ—9. Sterility clinic.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

THOMAS H. RUSSELL—9. Operations.
CARL EGGERS—2. Operations: Breast, head, and neck cases.
JOHN F. ERDMANN—2:30. Operations.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

HERBERT C. CHASE—11. Operations.
WILLIAM M. COOPER—2:30. Lecture and demonstration on varicose veins.

### PRESBYTERIAN HOSPITAL

ALLEN O. WHIPPLE and staff—9. Operations: Pancreas. Dry clinic and follow-up, pancreatic lesions.
DANA W. ATCHLEY, JOHN SCUDDER, and OCTA C. LEIGH—2. Symposium on fluid loss and fluid balance in surgery.
F. B. ST. JOHN, H. D. HARVEY, C. A. FLOOD, and ROSS GOLDEN—2. Symposium on gastric and duodenal lesions.

### ROOSEVELT HOSPITAL

CONDICT W. CUTLER, JR.—9. Gastric resection for carcinoma of the stomach.
ALFRED STILLMAN II—9. Resection of the colon for carcinoma.

### ST. FRANCIS' HOSPITAL

ROBERT B. LOBBAN and staff—9. Operations for primary hyperthyroidism, secondary hyperthyroidism, and nontoxic thyroid.

### ST. LUKE'S HOSPITAL

HENRY H. M. LYLE, JOHN DOUGLAS, EDWARD J. DONOVAN, WILLIAM F. MACFEE, MORRIS K. SMITH, and staff—9. Operative and dry clinics.
W. H. BERRY. Treatment of endocervicitis by electrocoagulation.
FREDERICK W. SOLLEY. Injection treatment of hydrocele.
H. J. SHELLEY. Injection treatment of varicose veins.
P. C. MORTON. Demonstration of rolling proctologic equipment table; relation of solitary polyps to carcinoma of the colon.
G. E. BURFORD. Problems in organization of a department of anesthesia.

### ST. VINCENT'S HOSPITAL

LOUIS F. SANMAN—2. Symposium: Surgical jaundice.
FRANCIS X. TIMONEY—3. Effect of iodine on the thyroid gland as seen in stage thyroidectomies.
ANTHONY ROTTINO—3. Pathological demonstrations.

## GENITO-URINARY SURGERY

### *Monday*

### HOSPITAL FOR JOINT DISEASES

PAUL W. ASCHNER, IRVING SIMONS, J. SIDNEY RITTER, and WILLIAM BISHER—2. Operations: Nephrectomy for tuberculosis, aseptic technique; pyelonephrolithotomy; pelvic ureterolithotomy. Dry clinics: Evaluation of excretion urography; cystometry and sphincterometry in diagnosis; seminal vesicles, diagnosis and therapy; selection of procedures in prostatic hypertrophy.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

DANIEL A. SINCLAIR—3:30. Operations.

### SYDENHAM HOSPITAL

RALPH L. DOURMASHKIN—2. Lesions in the female urethra; relationship of bone fractures to formation of calculi in the urinary tract; treatment of urethral calculi with rubber bags and metallic dilators.

*Tuesday*

FLOWER-FIFTH AVENUE HOSPITAL

SPRAGUE CARLETON and staff—9. Kidney and prostate surgery; demonstration of clinic apparatus, models and sketches.

MEMORIAL HOSPITAL

BENJAMIN S. BARRINGER—10. Suprapubic cystotomy for carcinoma.

METROPOLITAN HOSPITAL

SPRAGUE CARLETON and staff—1:30. Operations.

MORRISANIA CITY HOSPITAL

TERRY M. TOWNSEND—1:30. Operation: Prostatic obstruction.
JOHN DUFF—2. Dry clinic: Urological malignancies.
JOHN J. ROTH—2:30. Diverticula of the bladder.

NEW YORK CITY HOSPITAL

THOMAS J. KIRWIN—2. Operative and dry clinics.

NEW YORK HOSPITAL

ALEXANDER RAYMOND STEVENS, OSWALD TOWSLEY, and staffs—2. Operative and dry clinics.

NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

ANNE E. KUHNER—9. Dry clinic.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

JOSEPH A. HYAMS—2. Operative and dry clinics.

PRESBYTERIAN HOSPITAL

J. BENTLEY SQUIER and staff—2. Operations.

RIVERSIDE HOSPITAL

SIMON A. BEISLER—3. Urogenital conditions in the tuberculous.

UNITED STATES MARINE HOSPITAL

C. FERGUSON and R. A. MEE—2. Symposium on prostatic surgery.

WOMAN'S HOSPITAL

HENRY G. BUGBEE—2. Nephrectomy for pyonephrosis; genito-urinary problems in relation to gynecology and obstetrics.
ALBERT H. ALDRIDGE—2. Treatment of postoperative vesicovaginal fistula, demonstration of new technique, lantern slides.

*Thursday*

BELLEVUE HOSPITAL

ALEXANDER R. STEVENS and staff—9:30. Symposium on urinary calculus.
ALEXANDER R. STEVENS and staff—2. Operations.

BETH ISRAEL HOSPITAL

Staff—2:30. Operative and dry clinics.
ABRAHAM HYMAN. Differential diagnosis of renal and suprarenal tumors, with lantern slides.
SEYMOUR F. WILHELM. Diagnosis and operative treatment of male sterility, with lantern slides. Presentation of cases of unusual interest.

FLOWER-FIFTH AVENUE HOSPITAL

SPRAGUE CARLETON and staff—9. Kidney and prostate surgery; demonstration of clinical apparatus, models and sketches.

FRENCH HOSPITAL

CHARLES H. CHETWOOD and JOHN DENNIS GOONEY—9. Treatment of lacerated kidney and ureter, demonstration of cases; lacerations of bladder; lacerations of urethra; injuries to testes.

LINCOLN HOSPITAL

DAVID GEIRINGER, FRANCIS P. TWINEM, JORGE CAMPUZANO, and ISIDOR PALAIS—9. Operations.

NEW YORK CITY HOSPITAL

JOHN H. MORRISSEY—2. Perineal surgery.

NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

ANNE KUHNER—9. Dry clinic.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

F. JOSEPH MCCARTHY—3:30. Operations.

PRESBYTERIAN HOSPITAL

J. BENTLEY SQUIER and staff—2. Symposium on tumors of the kidney.

ROOSEVELT HOSPITAL

SIMON A. BEISLER—2. Transurethral prostatectomy; suprapubic prostatectomy; nephrectomy.

ST. LUKE'S HOSPITAL

Staff—10:30. Dry clinic.
HENRY H. M. LYLE. Ombredanne operation for hypospadias.
HENRY G. BUGBEE. Neoplasms of the urinary tract.
G. F. HOCH. Urinary calculi.
J. A. TAYLOR. Transurethral resection.
A. J. MURPHY. Urologic complications following hysterectomy.

*Friday*

FLOWER-FIFTH AVENUE HOSPITAL

SPRAGUE CARLETON and staff—9. Kidney and prostate surgery; demonstrations of clinical apparatus, models and sketches.

LENOX HILL HOSPITAL

Staff—9. Symposium on affections of the urinary tract and their treatment.
GEORGE W. SLAUGHTER. Use of sulfanilamide in urological infections.
HERBERT W. MEYER. Case presentations: Conservative surgery of strictures of the uretero-pelvic junction; repair of hypospadias—late result.
WILLIAM R. DELZELL. Perirenal air insufflation as an aid in diagnosis; motion pictures.
PETER A. NARATH. The rôle of the hydromechanics in the renal pelvis in general body infections.
HERBERT R. KENYON. The present status of transurethral prostatic resection.
MAXIMILIAN M. NEMSER. Presentation of interesting cases of renal tumors.

LUTHERAN HOSPITAL

TERRY M. TOWNSEND—2. Operations: Surgery of hypertrophied prostate.

METROPOLITAN HOSPITAL

SPRAGUE CARLETON and staff—1:30. Operations.

MISERICORDIA HOSPITAL

MAXIMILIAN NEMSER and HUBERT LYONS—9:30. Symposium on tumors of the urinary tract.
HUBERT LYONS. Diagnosis of renal, ureteral and vesical tumors.
MAXIMILIAN NEMSER. Management of tumors of the urinary tract; pyelographic changes in various types of renal tumors; lantern slides.
ROBERT C. SCHLEUSSNER—11. Pathologic classification of tumors of the urinary tract; demonstration of gross and microscopic specimens.

MOUNT SINAI HOSPITAL

GORDON D. OPPENHEIMER and MOSES SWICK—1:15. Operative and dry clinic.

NEW YORK POLYCLINIC MEDICAL SCHOOL

DAVID GEIRINGER—3:30. Operations.

PRESBYTERIAN HOSPITAL

J. BENTLEY SQUIER and staff—2. Operations.

ST. VINCENT'S HOSPITAL

HERBERT MOHAN, WENDELL J. WASHBURN, T. F. HOWLEY, E. CRAIG COATS, and G. J. MECCA—9. Operations.

SYDENHAM HOSPITAL

SAMUEL LUBASH—9. Operations: Plastic repair of the kidney pelvis in hydronephrosis.
SAMUEL MALISOFF—10:30. Operations: Transurethral resections for vesical neck obstructions.
LOUIS FRIEDMAN—1. Operations.

## OBSTETRICS AND GYNECOLOGY

### *Monday*

BETH ISRAEL HOSPITAL
(Jewish Maternity Hospital)

SAMUEL J. SCADRON and EDWIN G. LANGROCK—2. Obstetrical operations. Dry clinic: Cesarean section, demonstration of forceps delivery.

HARLEM HOSPITAL

HENRY C. FALK, PETER M. MURRAY, and MURRY H. LEVINE—2:30. Organization of gynecological service in the large city institution.
FREDERICK A. KASSEBOHM—3. Placenta praevia.

LYING-IN HOSPITAL

H. J. STANDER—2. Staff conference. Heart disease complicating pregnancy; dystocia in a patient with a contracted pelvis.

METROPOLITAN HOSPITAL

HORACE AYERS and staff—1:30. Dry clinic: Transplantation of ovaries, motion pictures, cardboard demonstration, presentation of patient.
DONALD E. BRACE and staff—1:30. Collection of placental blood for indirect transfusions, motion pictures.
L. S. LOIZEAUX and staff—1:30. Obstetrical and gynecological problems.

MOUNT SINAI HOSPITAL

SAMUEL H. GEIST and staff—1:30. Operative clinic: The treatment of prolapse, cystorectocele and incontinence in three typical cases.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

MORTIMER N. HYAMS and staff—2. Dry clinic: Uterosalpingography by fractional injections of lipiodol; sterilization of the female as an office procedure; conization of the uterine cervix.

PRESBYTERIAN HOSPITAL
(Sloane Hospital for Women)

W. W. HERRICK, ALVIN J. B. TILLMAN, and JEAN CORWIN—2. Symposium on the toxemias and other complications of pregnancy.

RIVERSIDE HOSPITAL

NELSON B. SACKETT—2. Gynecological complications in advanced pulmonary tuberculosis.

WOMAN'S HOSPITAL

WILLIAM T. KENNEDY—2. Complete abdominal hysterectomy for fibroids; operation for incontinence of urine, cystocele, rectocele, laceration of pelvic floor; demonstration of Kennedy technique and end results.
RALPH L. BARRETT—2. Electrocoagulation of cervix, two cases; demonstration of technique and end results.

### *Tuesday*

BELLEVUE HOSPITAL

E. W. HOLLADAY—9. Operations: Repair of old third-degree laceration.
WM. E. STUDDIFORD—9. Operations: Repair of cystocele and rectocele; suspension of uterus.
CLAUDE HEATON—9. Obstetrical ward rounds; complications of pregnancy; x-ray pelvimetry.
HOWARD C. TAYLOR, JR.—2. Care of patients suffering from gynecological malignancy.
WM. E. STUDDIFORD and HOWARD C. TAYLOR, JR.—3:30. Demonstration of pathological material.

BETH ISRAEL HOSPITAL

Staff—9. Gynecological dry clinic.
HERMAN LORBER. Carcinoma of fallopian tubes; carcinoma of ovary with peritoneal implants, five years after operation; early carcinoma of uterus.
MAURICE RASHBAUM. The diagnosis and management of urinary incontinence.
SEYMOUR WIMPFHEIMER. Chorioepithelioma.
E. A. HOROWITZ. The treatment of gonorrhea in women by systemic hyperpyrexia and simultaneous pelvic heating—an evaluation of 7½ years' experience. Rationale of treatment, indications, technique, apparatus required, results in lower genital tract infections, in salpingitis and in gonorrheal arthritis, late results; treatment of gonorrhea in women by sulfanilamide; fever therapy combined with pelvic heating; treatment of a female patient with gonorrhea.
MORTON VESELL. Dysgerminoma of ovary; granulosa cell tumor of ovary.
HERMAN LORBER and staff—2. Operations: Manchester operation for prolapse; vaginal hysterectomy; operation for incontinence.

FLOWER-FIFTH AVENUE HOSPITAL

HORACE AYRES and staff—2. Transplantation of ovaries; cardboard demonstration; motion pictures and presentation of patient.

DONALD BRACE—2. Collection of placental blood for indirect transfusions, motion pictures.
L. S. LOIZEAUX and staff—2. Advances in prenatal and postpartum care.
JOHN E. TRITSCH and staff—2. Demonstration of apparatus for collection and mensuration of postpartum blood. Results obtained from the use of several oxytocics on postpartum bleeding.

### FRENCH HOSPITAL

FREDERICK C. HOLDEN and staff—9. Pre-operative preparation of gynecological cases; nonoperative demonstration of LaForte operation for prolapse in elderly woman; Kennedy hysterectomy through vagina—nonoperative; discussion of extensive myomectomy with preservation of uterus in young woman; presacral resection of nerves for dysmenorrhea; bladder injuries in gynecological operations; postoperative care of gynecological patients.
F. C. HOLDEN, H. C. FALK, H. C. WILLIAMSON and staff—9. Gynecological and obstetrical operations; inspection of the new obstetrical hospital.

### HARLEM HOSPITAL

HENRY C. FALK, PETER M. MURRAY, and MURRY H. LEVINE—9. Operations; lecture on the tubal resection; a new and simple procedure for recurrent gonorrheal salpingitis.
JULIUS KURZROCK—3. Demonstration of forceps operation with special reference to the axis traction.

### HOSPITAL FOR JOINT DISEASES

ABRAHAM RONGY and staff—2. Interposition and other gynecological operations.
HOWARD E. LINDEMAN and staff—2. The Manchester and other gynecological operations.

### LENOX HILL HOSPITAL

PERCY H. WILLIAMS, ROBERT L. MCCREADY and staffs—9. Operations.

### LYING-IN HOSPITAL

B. H. GOFF—9. Modified LaForte operation for complete prolapse; operation for uterine prolapse.
Staff—2:30. Symposium on urinary tract infection of pregnancy.
HENRICUS J. STANDER. Relation of pyelitis to the toxemias of pregnancy.
C. M. MCLANE. Follow-up study of pyelitis of pregnancy.
H. F. TRAUT. Pyelonephritis as a complication of pyelitis of pregnancy.
R. C. BENSON. Postpartum and postoperative bladder retention and its treatment.
ROBERT G. DOUGLAS. Mandelic acid and sulfanilamide therapy in the treatment of pyelitis complicating pregnancy.
A. A. MARCHETTI. The pyelitis ileus syndrome.

### MEMORIAL HOSPITAL

WILLIAM P. HEALY—9. Gynecological operations.

### MOUNT SINAI HOSPITAL

SAMUEL H. GEIST and staff—9. Ward rounds and dry clinic; ovarian tumors; pathological and clinical discussion with demonstration of specimens; management of uterine bleeding.
ROBERT T. FRANK, MORRIS A. GOLDBERGER, and PAUL KLEMPERER—2. Exhibit of endocrine methods of diagnosis and hormone studies of cases.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

GERARD L. MOENCH—10. Influence of abnormalities of spermatozoa on sterility.
WALTER T. DANNREUTHER—2. Operations: Supravaginal hysterectomy for uterine fibroids; LaForte operation for complete prolapse; plication of the rectum and perineorrhaphy for enterocele; tracheloplasty for chronic endocervicitis.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

MALCOLM CAMPBELL—9. Gynecological operations.

### PRESBYTERIAN HOSPITAL
(Sloane Hospital for Women)

BENJAMIN P. WATSON and staff—9. Operations: Plastic for cystocele and rectocele; complete hysterectomy for cancer.
JAMES A. CORSCADEN—2. Cancer symposium.
E. S. COLER and J. H. BOYD—3:30. Demonstration of pathological material.

### ROOSEVELT HOSPITAL

HOWARD C. TAYLOR, THOMAS C. PEIGHTAL, HOWARD C. TAYLOR, JR., and W. E. ALSOP—9. Operations: Myomectomy; radium or hysterectomy for fibroids; radium or hysterectomy for postmenopausal bleeding.

### SYDENHAM HOSPITAL

JULIUS JARCHO—9. Operations.
ALFRED M. HELLMAN—10:30. Operations: Hysterectomy for fibroids and diseased adnexa; cesarean section (low flap).
JULIUS JARCHO—2. Roentgenography as an aid in obstetrical diagnosis.

### WOMAN'S HOSPITAL

EDWARD A. BULLARD—9. Operation for complete laceration of sphincter ani (silver wire); Sturmdorf operation for chronic endocervicitis and end results.
GEORGE G. BEMIS—9. Laparotomy for fibroids.

## *Thursday*

### BELLEVUE HOSPITAL

Staff—9. Dry Clinics.
ARTHUR REICH. Value of ergotrate in reducing incidence of postpartum hemorrhage.
SPENCER GURNEE. Sulfanilamide therapy in gonorrheal infections of female.
FRANCIS W. SOVAK. Operations for the relief of organic sterility in female, demonstration of successful cases.
WM. E. STUDDIFORD. Treatment of incomplete abortion complicated by fever.
M. GOLDBLATT and L. LANGMAN. Analysis of 342 cases operated upon for ectopic pregnancy.
IRA I. KAPLAN—2. Operation: Application of radium for carcinoma of cervix.
FRANCIS W. SOVAK—2. Operation: Reconstruction of fallopian tubes.
DAVID N. BARROWS—2. Operation: Supravaginal hysterectomy for fibromyomata uteri.

### FLOWER-FIFTH AVENUE HOSPITAL

HENRY B. SAFFORD and staff—9. Gynecological and obstetrical surgery.

### HARLEM HOSPITAL

HENRY C. FALK, PETER M. MURRAY, and MURRY H. LEVINE—9. Classification and management of the infected abortion; operations.

A. CHARLES POSNER—3. Breach presentation; Methods employed to reduce foetal and neonatal mortality.

### LINCOLN HOSPITAL

EDWARD J. DAVIN and staff—9. Use of intravenous ergonovine at the end of the second stage of labor, results in over 500 cases.

HAROLD C. INGRAHAM—2. Treatment of pelvic inflammatory disease by various modalities.

ROBERT L. CRAIG—2. Treatment of pelvic inflammatory disease by acetyl-beta-methyl-cholin.

FRANK SPIELMAN—2. Gynecological pathology, some unusual ovarian neoplasms.

### LUTHERAN HOSPITAL

JAMES T. PADGETT—9. Operations.

### LYING-IN HOSPITAL

HENRICUS J. STANDER—9. Operation for cure of cystocele and rectocele.

HERBERT F. TRAUT—9. Myomectomy and hysterectomy.

HENRICUS J. STANDER—2:30. A study of acute yellow atrophy.

HERBERT F. TRAUT—3. The clinical and histological differentiation between hypertrophy and hyperplasia of the endometrium.

JOHN B. PASTORE—3:30. Anemia in pregnancy.

ROBERT G. DOUGLAS—4. Intrapartum infection and its relation to the time of cesarean section.

### METROPOLITAN HOSPITAL

L. S. LOIZEAUX—9. Operations.

### MISERICORDIA HOSPITAL

JAMES P. HENNESSY—9:30. Modified Scanzoni for occiput posterior.

F. WALTER GRAVELLE—10. Forceps technique; single application for occiput posterior.

ALEXANDER H. SCHMITT—10:30. Complications requiring cesarean section.

FREDERICK E. NEEF—2. Application of radon ring in carcinoma of the uterine cervix.

JOSEPH A. DEVLIN—3. Operative indications in gynecological emergencies.

FRANCIS W. SOVAK—4. Operative treatment for sterility; lantern slides.

### MOUNT SINAI HOSPITAL

ISIDOR C. RUBIN—9:30. Ward rounds and demonstration of cases: Genital tuberculosis, kidney tumors, myomectomy, vaginal supravaginal hysterectomy and parametrial fixation for prolapse and myoma.

ISIDOR C. RUBIN and staff—2. Operations: Parametrial fixation (Donald Fothergill) for prolapse of uterus, local analgesia; multiple myomectomy (show hippuran); laparotomy for salpingolysis; plastic for cystorectocele, parasacral anesthesia; motion picture of parametrial fixation.

### NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

WILHELMINA A. RAGLAND and associates—9. Toxemias of pregnancy; x-ray pelvimetry; care of prematures.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

LOUIS J. LADIN—9. Operations.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

THEODORE NEUSTAEDTER and staff—10. Clinical use of estrogenic hormones.

ADOLPH JACOBY and staff—2. Treatment of pelvic exudates with iontophoresis.

### PRESBYTERIAN HOSPITAL
(Sloane Hospital for Women)

BENJAMIN P. WATSON and staff—9. Cesarean sections; obstetrical rounds; cardiac, toxemias and other complications.

W. E. CALDWELL, HOWARD C. MOLOY, and D. ANTHONY D'ESOPO—2. Diagnosis of pelvic abnormality; x-ray pelvimetry; the precision stereoscope in the mechanism of labor.

### ROOSEVELT HOSPITAL

HOWARD C. TAYLOR, THOMAS C. PEIGHTAL, HOWARD C. TAYLOR, JR., and WILSON E. ALSOP—9. Operations: Kennedy operation for bladder incontinence: operation for cystocele and rectocele; tubal implantation of occluded tubes.

THOMAS C. PEIGHTAL, HOWARD C. TAYLOR, JR., WILSON E. ALSOP, and A. V. GREELEY—2. Dry clinic: Pelvic cancer; postmenopausal bleeding; endometriosis; tubal implantation cases; demonstration of ovarian tumors.

### ST. FRANCIS' HOSPITAL

CHARLES VEJVODA and staff—9. Subtotal hysterectomy; total hysterectomy; salpingectomy, uterine suspension.

### SYDENHAM HOSPITAL

JULIUS JARCHO—9. Operations.

ALFRED M. HELLMAN—2. Dry clinic: Large mesenteric tumor; cornual pregnancy; Kennedy hysterectomy; presentation of patients and specimens; skiodan salpingography.

### WOMAN'S HOSPITAL

RALPH A. HURD—9. Cesarean section, low flap.

ALBERT H. ALDRIDGE—9. Cesarean section, Latzko; demonstration of modified technique and end results, with lantern slides; vaginal hysterectomy for uterine prolapse, Bissell technique.

DR. CARY—9. Treatment of sterility, demonstration of spermatozoa, motion pictures.

NELSON B. SACKETT—2. Abdominal hysterectomy for carcinoma of corpus uteri, postradiation.

GEORGE GRAY WARD—2. Insertion of radium for carcinoma of cervix; end results of treatment of gynecological cancer, demonstration of treated cases.

HARRIET MCINTOSH—2. X-ray therapy for gynecological cancer.

## *Friday*

### BELLEVUE HOSPITAL

E. W. HOLLADAY—9. Operation: Repair of cystocele and rectocele.

HOWARD C. TAYLOR, JR.—9. Laparotomy for ovarian cyst; complete hysterectomy for carcinoma of corpus uteri.

I. WELLEN—9. Obstetrical ward rounds; toxemias of pregnancy.

A. REICH—9. Demonstration of Barton forceps.

### FLOWER-FIFTH AVENUE HOSPITAL

HORACE AYERS and staff—9. Operations.

### HARLEM HOSPITAL

FREDERICK A. KASSEBOHM, JULIUS KURZROCK, and A. CHARLES POSNER—3. Emergency operative clinic.

### LENOX HILL HOSPITAL

ROBERT L. MCCREADY, PERCY H. WILLIAMS, and staffs—9. Operations.

### LINCOLN HOSPITAL

JOHN A. KELLEY—9. Treatment of carcinoma of cervix and corpus uteri.

### LUTHERAN HOSPITAL

FREDERICK A. KASSEBOHM—9. Emergency obstetrical procedures.

### LYING-IN HOSPITAL

HERBERT F. TRAUT and ROBERT G. DOUGLAS—9. Demonstration of the organization and operation of a cancer follow-up clinic.

### METROPOLITAN HOSPITAL

HENRY B. SAFFORD and staff—9. Operations.

### MOUNT SINAI HOSPITAL

ISIDOR C. RUBIN and staff—9:30. Sterility clinic: Mechanical aspects of tubal insufflation; motion pictures; endocrine aspects in outpatient department.

SAMUEL H. GEIST and staff—2. Some aspects of gynecological urology.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

THOMAS H. CHERRY—9. Operations: Anterior colporrhaphy and perineorrhaphy; vaginal hysterectomy for uterine fibroids; plastic repair of fallopian tubes for sterility; tracheloplasty for chronic endocervicitis.

WALTER T. DANNREUTHER—10. Diagnosis of ectopic pregnancy, lantern slide demonstration.

WALTER T. DANNREUTHER—2. Operations: Radium application for carcinoma of the cervix; oöphorectomy for ovarian tumor; interposition operation for complete prolapse; supravaginal hysterectomy for uterine fibroids.

I. W. KAHN and staff—3. Important urological conditions in gynecological patients.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

DAVID N. BARROWS—9. Gynecological operations.

EDWARD H. DENNEN—10. Obstetrical problems, lecture.

### PRESBYTERIAN HOSPITAL
(Sloane Hospital for Women)

B. P. WATSON and staff—9. Operations; obstetrical rounds; obstetrical analgesia and anesthesia.

RAPHAEL KURZROK and associates—2. Symposium on endocrine disturbances.

### ROOSEVELT HOSPITAL

HOWARD C. TAYLOR, THOMAS C. PEIGHTAL, HOWARD C. TAYLOR, JR., and WILSON E. ALSOP—9. Fothergill operation for uterine prolapse; operation for endometriosis; operation for chronic adnexal disease.

### ST. FRANCIS' HOSPITAL

ALEXANDER NICOLL and staff—9. Hysterectomy; total hysterectomy; subtotal salpingectomy and suspension.

### ST. VINCENT'S HOSPITAL

WILLIAM M. FORD, WALLACE KRUGLER, JOHN F. MCGRATH, JAMES P. HENNESSY, and JOSEPH E. CORR—9. Operations.

### SYDENHAM HOSPITAL

JULIUS JARCHO—2. Dry clinic: Uterosalpingography—visualization of the internal genitalia by means of injection of contrast media.

### WOMAN'S HOSPITAL

EDWARD A. BULLARD—9. Operation for cystocele, rectocele, laceration of pelvic floor.

ALBERT H. ALDRIDGE—9. End results of spontaneous versus prophylactic methods of delivery, with lantern slides.

JOSHUA W. DAVIES—9. Operation for incontinence of urine, cystocele, rectocele, laceration of pelvic floor.

HARRIET MCINTOSH—9. X-ray pelvimetry, Caldwell-Moloy technique.

ARTHUR J. MURPHY—2. Operation for fibroids.

GEORGE GRAY WARD—2. Vaginal hysterectomy for uterine prolapse, Mayo technique.

DR. WHITE—2. Diagnosis and treatment of intestinal obstruction in gynecological cases.

## NEUROSURGERY

### *Monday*

### NEUROLOGICAL INSTITUTE

BYRON STOOKEY—2. Operation for brain tumor.

### NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

JOSEPH E. J. KING—2:30. Operations.

### *Tuesday*

### BELLEVUE HOSPITAL

JOSEPH E. J. KING—2. Operations.

Staff—2. Symposium on neurological surgery.

W. D. WINGEBACH. Cases of brain and spinal cord tumors.

JOSEPH E. J. KING. Demonstration of cases: Brain abscess, osteomyelitis of the skull, meningiomata of the middle fossa.

ABRAHAM KAPLAN. Operation for meningioma of the brain, motion pictures; cases of extradural and subdural hematomata.

FRANK TURNEY. Case of subdural hygroma; ventriculography and encephalography; Munro's tidal drainage.

### NEUROLOGICAL INSTITUTE

CLARENCE HARE and GABRIEL SCHWARTZ—9:30. Carcinoma of the lung metastases to the brain.

GEORGE HYSLOP—9:30. Thyroid metastases to the brain.

BYRON STOOKEY—9:30. Neurosurgical measures for relief of intractable pain due to metastatic carcinoma.

*Thursday*

MONTEFIORE HOSPITAL

IRA COHEN—2. Operations.

MOUNT SINAI HOSPITAL

IRA COHEN and A. KAPLAN—9. Operations.

NEUROLOGICAL INSTITUTE

FRITZ CRAMER—9:30. Relative occurrence of convulsive seizures in depressed fractures of the skull—elevated and non-elevated.

KATE CONSTABLE—9:30. Relative occurrence of convulsive seizures following concussion without cranial fracture.

JOHN E. SCARFF—9:30. Cerebral scars in convulsive states and their treatment.

NEW YORK HOSPITAL

GEORGE J. HEUER and staff—2. Dry clinics: Surgery of the nervous system.

GEORGE J. HEUER. Surgery of the hypophysis.

BRONSON S. RAY. Direct radiation of brain and spinal cord tumors on the operating table.

HAROLD G. WOLFF. Headache.

JOSEPH C. HINSEY. Visceral pain.

N. CHANDLER FOOT. Tumors of the peripheral nerves.

*Friday*

MOUNT SINAI HOSPITAL

IRA COHEN and A. KAPLAN—9. Operations.

NEUROLOGICAL INSTITUTE

CLEMENT MASSON—9:30. Tumors of the brain in children.

BYRON STOOKEY—9:30. Tumors of the cord in children.

## FRACTURES AND TRAUMATIC SURGERY

*Monday*

HARLEM HOSPITAL

LOUIS T. WRIGHT, RALPH H. YOUNG, DAVID H. SMITH, CALDWELL B. ESSELSTYN, JESSE J. GREENE, A. ZINGARO, MAURICE C. O'SHEA, FRANCIS L. MORHARD, and THOMAS C. CASE—3:15. Fracture symposium.

PRESBYTERIAN HOSPITAL

WILLIAM DARRACH, CLAY R. MURRAY and staff—2. Dry clinic.

*Tuesday*

BEEKMAN STREET HOSPITAL

Staff—9. Ward rounds and dry clinics.

ROBERT H. KENNEDY. Fracture of shaft of the femur.

JAMES H. HEYL. Fractures of the elbow.

SIGMUND MAGE. Compound fractures.

LESTER BLUM. Fractures of the shaft of the humerus.

ROBERT T. FINDLAY. Conservative treatment versus immediate amputation in severe injuries of the hand and forearm.

THOMAS M. LOWRY. Fractures of the scaphoid.

MYRON A. SALLICK. Russell traction.

BELLEVUE HOSPITAL

J. GORDON LEE, HERBERT M. BERGAMINI, ROBERT P. WADHAMS, KENNETH M. LEWIS, and GEORGE A. KOENIG—12:30. Symposium on fractures, return clinic, ward rounds following clinic.

FLOWER-FIFTH AVENUE HOSPITAL

MILTON J. WILSON and staff—9. Demonstration of Russell traction and other fracture problems.

MISERICORDIA HOSPITAL

SAMUEL B. BURK—9:30. Operative and dry clinics.

FRANCIS X. TIMONEY—11. Diagnosis and treatment of fractures.

NEW YORK CITY HOSPITAL

PRESTON WADE and BOARDMAN BOSWORTH—9. Operative and dry clinics.

LYMAN W. CROSSMAN, DAVID SLOANE, and CHARLES M. GRATZ—9. Intrapelvic protrusion of the acetabulum (Otto pelvis); tendon transplantation for wrist drop caused by posterior interosseous nerve paralysis following a compound fracture of the neck of the radius; cases illustrating various fractures of knee joint; traumatic dislocations; neurotrophic joints.

PRESBYTERIAN HOSPITAL

WILLIAM DARRACH, CLAY R. MURRAY, and staff—9. Operations.

WILLIAM DARRACH, CLAY R. MURRAY, and staff—2. Follow-up clinic.

ST. VINCENT'S HOSPITAL

CONSTANTINE J. MACGUIRE, JR.—2. Injuries of the carpal bones, treatment.

WILLIAM G. DORAN—2. Healing of fractures with acute hematogenous osteomyelitis; immediate treatment of compound fractures; institutional treatment of fractures; treatment of compound dislocation of ankle joint.

PRESTON A. WADE—2. Fractures of the neck of the femur; comparison of results; Whitman abduction method with internal fixation.

*Thursday*

COLUMBUS HOSPITAL

FILIPPO CASSOLA—9:30. X-ray traumatological exhibit.

FRENCH HOSPITAL

SETH M. MILLIKEN, ROBERT P. WADHAMS, and associates—9. Traction suspension treatment of fractures of the clavicle, femur, tibia and fibula; skeletal traction versus skin traction in fractures; demonstration of Kirschner wire and Steinman pin; bilateral fracture of os calcis and fifth metatarsal treatment; treatment of fractured skulls; fracture dislocation of cervical vertebra, late reduction; refracture of tibia and fibula 18 months after repair with sliding graft; conservation of useful hand following traumatic avulsion of fingers and flexor tendons; rupture of liver with recovery; rupture of spleen with recovery; x-ray demonstration by Elmer M. Claiborne.

KNICKERBOCKER HOSPITAL

GEORGE A. KOENIG, PHILIP D. ALLEN, RICHARD J. O'CONNELL, EDWARD R. EASTON, D. REES JENSEN, and WADE DULEY—1. Fracture of the shaft of the humerus in the

aged; follow-up of fracture of the neck of the femur treated by internal fixation; supracondylar fracture of the humerus in children; compound comminuted fracture of the tibia and fibula; dislocation of the femoral head; anterior dislocation of the cervical vertebræ; fracture of the lumbar vertebræ; divergent fracture dislocation of the metatarsal bones.

LENOX HILL HOSPITAL

Staff—9. Fracture Clinic.
WALTER I. GALLAND. Ambulatory skeletal traction for overriding fractures of clavicle; dislocations of the carpal semilumar bone, 2 cases; reduction with the aid of the Thomas wrench.
JOHN C. A. GERSTER. Open reduction of fractures.
ALFRED F. WIESENTHAL. Fractures of the spine.
HENRY JORDAN. After care of fractures with special reference to brace treatment.
SIDNEY GAYNOR. Ischial seat brace; Lorenz osteoclast for treatment of fractures; a cabinet for storing plaster of Paris bandages.
Inspection of hospital brace shop.

LINCOLN HOSPITAL

KIRBY DWIGHT, EDWARD D. TRUESDELL, JAMES R. LINCOLN, and JACOB FRIEDMAN—2. Symposium on fractures. Ward rounds.

METROPOLITAN HOSPITAL

MILTON J. WILSON and staff—1:30. Russell traction treatment of compound fractures.

MORRISANIA CITY HOSPITAL

EUGENE J. BOZSAN—9:30. Obscure traumatisms of the hipjoint.
THOMAS I. BRENNAN—9:30. Operations.
THOMAS J. O'KANE and FREDERICK W. WILLIAMS—11. Diabetic surgery of the extremities.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

HERBERT M. BERGAMINI and EMMETT A. DOOLEY—2. Operative and dry clinics: Fractures of the neck of the femur. (At Reconstruction Hospital.)
JOHN J. MOORHEAD—2. Operations.
H. H. RITTER—2. Operations.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

DAVID M. BOSWORTH—10. Operative and dry clinics.

PRESBYTERIAN HOSPITAL

WILLIAM DARRACH, CLAY R. MURRAY, and staff—9. Operations.
WILLIAM DARRACH, CLAY R. MURRAY, and staff—2. Traction clinic.

UNITED STATES MARINE HOSPITAL

E. C. STAMM and staff—10:30. Symposium on fractures of the jaw.
F. LIBERSON and S. P. COOPER—2. Symposium: Shoulder injuries, x-ray aspects and follow-up clinics.
Staff—2. Symposium on head injuries.
HAROLD KELMAN. Neurological aspects.
L. A. PALMER and staff. Surgical aspects.

*Friday*

BEEKMAN STREET HOSPITAL

Staff—9. Ward rounds and dry clinics.
ROBERT H. KENNEDY. Fracture of shaft of the femur.
JAMES H. HEYL. Fracture of the elbow.
SIGMUND MAGE. Compound fractures.
LESTER BLUM. Fractures of the shaft of the humerus.
ROBERT T. FINDLAY. Conservative treatment versus immediate amputation in severe injuries of the hand and forearm.
THOMAS M. LOWRY. Fractures of the scaphoid.
MYRON A. SALLICK. Russell traction.

FLOWER-FIFTH AVENUE HOSPITAL

MILTON J. WILSON and staff—9. Treatment of compound fractures; demonstrations of end results.

PRESBYTERIAN HOSPITAL

WILLIAM DARRACH, CLAY R. MURRAY, and staff—9. Fracture conference.

## PLASTIC AND FACIOMAXILLARY SURGERY

*Tuesday*

LINCOLN HOSPITAL

GUSTAV AUFRICHT and JACOB FRIEDMAN—2. Operative and dry clinics.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

GUSTAV AUFRICHT and staff—9. Operative and dry clinics.
FRED SQUIER DUNN—10:30. Operations.

PRESBYTERIAN HOSPITAL

JEROME P. WEBSTER and THOMAS W. STEVENSON, JR.—9. Operations.

SYDENHAM HOSPITAL

THEODOR BLUM—10. Ameloblastoma from the clinical x-ray and pathologic standpoint with presentation of cases.
JACQUES W. MALINIAC—1:30. Operations.

*Thursday*

BETH ISRAEL HOSPITAL

ARTHUR J. BARSKY—9. Operations.

LUTHERAN HOSPITAL

KEITH KAHN—2. Operations.

NEW YORK CITY HOSPITAL

ALEXANDER ZIMANY—9. Operative and dry clinics.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

HAROLD S. VAUGHAN and staff—11. Operations.

PRESBYTERIAN HOSPITAL

JEROME P. WEBSTER and THOMAS W. STEVENSON, JR.—9. Operations.
HENRY S. DUNNING—2. Operations: Harelip; cleft palate; bone graft of mandible; ankylosis of temporo-mandibular joint.

SYDENHAM HOSPITAL

JACQUES W. MALINIAC—3. Dry clinic.

*Friday*

BELLEVUE HOSPITAL

YOLANDE H. HUBER—9:30. Operations.

YOLANDE H. HUBER—2. Technique of dressings used for free transplants, moulages; cases demonstrating Wolffe grafts; cases of reconstruction for deformities following burns of face, hands and feet, demonstrating use of pedicled grafts; breast reconstructions, usual type; breast reconstructions with free nipple transplants; rhinoplasties and reconstructions for nasal defects; temporal muscle transplant for paralysis of the facial nerve; miscellaneous plastic and reconstructive cases; demonstrations with lantern slides and motion pictures; "side show," moulage, etc. Operation: Breast reconstruction.

KNICKERBOCKER HOSPITAL

CLARENCE R. STRAATSMA—9. Plastic operations on the face.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

CLARENCE R. STRAATSMA and staff—9. Operative and dry clinics.

PRESBYTERIAN HOSPITAL

JEROME P. WEBSTER and THOMAS W. STEVENSON, JR.—2. Dry clinic: Congenital and acquired deformities of nose, ears, lips, face, chin, body, extremities and penis; special conditions, including osteomyelitis, hemangiomata, keloids, pigmented moles, melanomata, Kaposi's disease, epitheliomata, fibrosarcomata, syphilis, lupus and tattoes.

## THORACIC SURGERY

*Monday*

BELLEVUE HOSPITAL

JAMES A. MILLER, J. BURNS AMBERSON, A. V. S. LAMBERT, and FRANK B. BERRY—2. Symposium on diseases of the chest.

FLOWER-FIFTH AVENUE HOSPITAL

SAMUEL THOMPSON—2. Operations.

JOHN S. O. HERRLIN—2. Acute empyema.

METROPOLITAN HOSPITAL

SAMUEL THOMPSON and staff—1:30. Operative and dry clinics.

MONTEFIORE HOSPITAL

ARTHUR H. AUFSES and ISIDOR KROSS—2. Operations.

ST. VINCENT'S HOSPITAL

FRANK MURRAY and DANIEL A. MULVIHILL—3. Treatment of lung abscess, medical and surgical aspects.

*Tuesday*

MOUNT SINAI HOSPITAL

HAROLD NEUHOF, ARTHUR S. W. TOUROFF, and AMEIL GLASS—1:15. Operations.

HAROLD NEUHOF, COLEMAN B. RABIN, HERMAN HENNELL, DR. BURLINER, AMEIL GLASS, and ARTHUR S. W. TOUROFF—1:15. Symposium on abscess of the lung: Pathogenesis, pathology, clinical features, roentgenological features, localization, operative treatment, results of operation.

NEW YORK CITY HOSPITAL

RICHMOND L. MOORE and G. H. HUMPHREYS—9. Operative and dry clinics.

LYMAN W. CROSSMAN and staff—2. Surgical aspects of bronchiectasis; lantern slide demonstration with motion picture of a lobectomy in color.

SYDENHAM HOSPITAL

ARTHUR S. UNGER, MILTON FRIEDMAN, J. TOWNSEND TRAVERS, SOLOMON FINEMAN, and DAVID E. EHRLICH—10. Symposium: Diagnosis and treatment of lung tumors.

*Thursday*

BELLEVUE HOSPITAL

A. V. S. LAMBERT—9:30. Operations.

LENOX HILL HOSPITAL

Staff—2. Symposium on non-tuberculous pulmonary suppuration.

H. MCLEOD RIGGINS. Etiology of bronchiectasis.

GRANT THORBURN. Medical treatment of bronchiectasis.

JOHN D. KERNAN. Bronchoscopic treatment of bronchiectasis.

CARL EGGERS. Surgical treatment of bronchiectasis.

WM. H. STEWART. Roentgen diagnosis and treatment of non-tuberculous intrapulmonary suppuration.

GIRARD F. OBERRENDER. The rôle of foreign bodies in the etiology of intrapulmonary suppuration.

WALTER T. STENSON. The surgical treatment of lung abscess.

GEORGE MUEHLECK. Intrapulmonary suppuration complicated by empyema.

OTTO C. PICKHARDT. The association of pulmonary suppuration with carcinoma of the lung.

MEMORIAL HOSPITAL

Staff—2. Symposium on tumors of lung and mediastinum.

LLOYD F. CRAVER. Diagnosis of interesting lung tumors.

J. SAMUEL BINKLEY. Demonstration of the aspiration biopsy technique in lung tumors.

EDWARD ELLIS. Demonstration of the tumor material obtained by aspiration biopsy in lung tumors.

METROPOLITAN HOSPITAL

SAMUEL A. THOMPSON—1:30. Operations.

NEW YORK HOSPITAL

GEORGE J. HEUER and staff—9. Operative and dry clinics.

GEORGE J. HEUER. Surgical treatment of pulmonary suppuration.

WILLIAM DEW. ANDRUS. Surgery of mediastinal tumors.

RALPH F. BOWERS. Acute and chronic empyema.

CRANSTON W. HOLMAN. Scoliosis in intrathoracic disease.

EDGAR MAYER. Collapse therapy in pulmonary tuberculosis.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

LOUIS R. DAVIDSON—2. Operative and dry clinics.

PRESBYTERIAN HOSPITAL

RICHMOND L. MOORE, D. W. RICHARDS, JR., BYRON STOOKEY, ROSS GOLDEN, and JOHN D. KERNAN—2. Symposium on tumors of the lung.

*Friday*

LENOX HILL HOSPITAL

Staff—2. Symposium on surgical treatment of pulmonary tuberculosis.
GRANT THORBURN. Indications for collapse therapy.
PHILIP G. C. BISHOP. Pneumothorax.
HERBERT C. MAIER. Intrapleural pneumolysis.
WALTER T. STENSON. Possible pitfalls in phrenic nerve crushing.
HERBERT W. MAYER. Results of surgical treatment of pulmonary tuberculosis at Lenox Hill Hospital during the last twenty years.
OTTO C. PICKHARDT. Extrapleural thoracoplasty.
H. MCLEOD RIGGINS. Tuberculous empyema—medical treatment.
CARL EGGERS. Tuberculous empyema—surgical treatment.
JOHN D. KERNAN, GIRARD OBERRENDER and H. MCLEOD RIGGINS. Tuberculosis of the bronchi.
WILLIAM H. STEWART. Cinefluorographic demonstration of respiration: Breathing in normal cases; breathing in patients with pulmonary tuberculosis; breathing in patients after phrenic crushing; breathing in patients with pneumothorax; breathing in patients after extrapleural thoracoplasty.

RIVERSIDE HOSPITAL

LOUIS CARP—9. Operations: Phrenemphraxia; pneumonolysis; thoracoplasty.
LOUIS CARP and staff—2. Dry clinics.
LOUIS CARP. Cold abscess of chest wall, thoracoscopy and contra-indications for pneumonolysis.
ARTHUR H. AUFSES. Indications for thoracoplasty.
JEROME M. ZIEGLER. The management of tuberculous empyema.
BORRIS A. KORNBLITH. Follow-up results in phrenemphraxia.
MAX TASCHMAN. Demonstration of pneumothorax and its complications.

ROOSEVELT HOSPITAL

FRANK B. BERRY—9. Lobectomy and thoracoplasty.

ST. LUKE'S HOSPITAL

A. E. W. ADA and associates—9. Operative and dry clinics.
P. G. C. BISHOP. Selective bronchography.
S. T. ALLISON. Diseases of the mediastinum.
O. R. JONES. Pneumothorax therapy for tuberculosis during pregnancy.
L. C. KNOX. Pathology of primary carcinoma of the lung.
A. E. W. ADA. Surgical treatment of acute abscess of the lung.

## SURGERY OF THE BONES AND JOINTS

*Monday*

BELLEVUE HOSPITAL

ARTHUR KRIDA—2. Operations.

HOSPITAL FOR JOINT DISEASES

HARRY FINKELSTEIN and staff—2. Dry clinic: Cases of leg lengthening; rachitic deformities treated by decalcification method; stenosing tendovaginitis; epiphyseolysis of the hip; synostosis operation for ununited fractures of the tibia and fibula; cases of hallux valgus (new operation); cases of subtrochanteric osteotomy by the inverted "L" procedure; low back lesions; venography for muscle angiomata; glomus tumors.

METROPOLITAN HOSPITAL

ANSON H. BINGHAM and staff—1:30. Operative and dry clinics.

MISERICORDIA HOSPITAL

GASTON A. CARLUCCI—2. Osteomyelitis—the surgical problem.

NEW YORK ORTHOPAEDIC HOSPITAL

HALFORD HALLOCK—1:30. General clinic, including all orthopedic conditions.

*Tuesday*

HOSPITAL FOR JOINT DISEASES

LEO MAYER and staff—9. Dry clinic.
LEO MAYER. Cases illustrating reconstructive tendon surgery.
NICHOLAS S. RANSOHOFF. Abdominal fascial transplants for abdominal paralysis.
BENJAMIN WOLFORT. Adamantinoma of the tibia.
JOSEPH MILGRAM. The aspiration therapy of subacromial bursitis.
DAVID TELSON. Fractures of the neck of the femur treated by wiring.
HARRY D. SONNENSCHEIN and staff—2. Dry clinic.
HARRY D. SONNENSCHEIN. Two cases of resection of the elbow joint; lipoid granulomatosis of ribs and sacrum.
MICHAEL S. BURMAN. Complete condylar fracture of the humerus in an adult; operative removal of displaced condyles; tourniquet paralysis of lower extremity following knee operation; operative release of sciatic nerve; two cases of fracture of posterior arch of atlas.
JOSEPH G. WISHNER. Two cases of congenital dislocation of hips diagnosed soon after birth; subcutaneous drilling for the relief of pain in osteo-arthritis of the hip after old slipped epiphysis of the upper femoral epiphysis.
MARK M. YOUMANS. Three cases of giant cell tumor of the spine.
ABRAHAM KENIN. Case of scoliosis treated by wedged turnbuckle jacket and two-stage spine fusion operation; case of Scheuermann's disease of the spine; case of narrowed intervertebral disc between "$L_5$" and "$S_1$."
SAUL RITCHIE. A complicated wrist fracture—comminuted Colles' fracture; fracture of the scaphoid and fracture with displacement of the os magnum; operative removal of displaced head: fracture of the neck of the femur in a child; slipped upper femoral epiphysis fracture of the neck of the femur in reduction, end result.

LOUIS SALTZMAN. Operative removal of free bodies in peroneal tendon sheath.
ADOLPH A. SCHMIER. Fracture dislocation of the elbow—2 cases—one with median nerve involvement and one with ulnar nerve involvement.
ISADORE ZADEK and staff—2. Operations: Horizontal wedge arthrodesis for flat foot.

### MONTEFIORE HOSPITAL

SETH SELIG—10. Dry clinic: Diagnosis and treatment of unsuspected adrenal insufficiency in bone tuberculosis.
ROBERT K. LIPPMAN—10. Dry clinic: Bone tuberculosis.

### NEW YORK ORTHOPAEDIC HOSPITAL

Staff—9. Symposium on scoliosis.
ALAN DEF. SMITH. Routine treatment; organization of special clinic; criteria for selection of patients for operation; compensation.
ALBERT B. FERGUSON. Measurement of the curve; identification of primary curve; determination of area for operation.
MCDOWELL ANDERSON. The jacket; principles, application and preparation for operation; semi-bent and straight jacket.
BENJAMIN P. FARRELL. Spine fusion operation technique.
ALAN DEF. SMITH. Results of operative treatment.
GEORGE L. INGE—1:30. Scoliosis clinic: Demonstration of cases before and after operation.

### HOSPITAL FOR RUPTURED AND CRIPPLED

PHILIP D. WILSON and EARL E. VAN DERWERKER—9. Ward rounds, children's orthopedic service.
Staff—10. End result clinic.
Staff—10:30. Regular weekly orthopedic staff meeting, with service reports from house surgeons.
RAYMOND W. LEWIS. X-ray reports.
DOMINIC A. DESANTO. Interesting pathological specimens.
BRADLEY L. COLEY, NORMAN L. HIGINBOTHAM, and DOMINIC A. DESANTO—2. Discussion of problems in diagnosis and treatment of bone tumors.

### ST. LUKE'S HOSPITAL

MATHER CLEVELAND and DAVID M. BOSWORTH—2. Operative and dry clinics: Tibial distraction; treatment of fracture of the neck of the femur.

## *Thursday*

### BELLEVUE HOSPITAL

ARTHUR KRIDA and staff—2. Symposium on orthopedic surgery: Intracapsular fracture of neck of the femur, bone peg operation; epiphyseal fracture of the neck of the femur, bone peg operation; congenital dislocation of the hip, genu recurvatum operation; cruciate ligament repair; encircling fascial band operation for splay foot and hallux valgus; congenital absence of tibia; Charcot's knees; fusion operation; modified reconstruction of the hip for osteo-arthritis.

### FLOWER-FIFTH AVENUE HOSPITAL

A. H. BINGHAM and staff—9. Problems in orthopedic surgery; demonstration of cases, illustrated by motion pictures and lantern slides.

### GOUVERNEUR HOSPITAL

WALTER D. LUDLUM, JR., JOHN P. STUMP, and staffs—2. Presentation of cases from orthopedic and traumatic services.
JOHN P. STUMP. Fracture of the femoral neck, conservative treatment, three cases. Flexion-adduction deformity of hip, operative correction.
SYDNEY NARINS. Lumbar puncture needle injury of an intervertebral disc.
WALTER D. LUDLUM, JR. Slipped capital femoral epiphysis, open reduction, two cases.
MILES C. KREPELA. Fracture of shaft of humerus, traction-suspension treatment, three cases.
CARL A. PETERSON. Head injuries with visual disturbances, two cases.
DANTE P. DAPOLONIA. Fractured femur in children, Russell traction treatment, three cases.

### HOSPITAL FOR JOINT DISEASES

SAMUEL A. JAHSS and staff—9. Operations.
HARRY FINKELSTEIN and staff—9. Operations: Leg lengthening; stenosing tendovaginitis; correction of rachitic deformities; new operation for hallux valgus; reduction of epiphyseolysis of the hip.
SAMUEL A. JAHSS and staff—2. Dry clinic.

### MOUNT SINAI HOSPITAL

Staff—9. Operative and dry clinics.
SETH SELIG. Fixation of osteo-arthritis of the hip joint by Smith-Petersen nail.
ROBERT LIPPMANN. Internal fixation of intracapsular fracture of the neck of the femur by the corkscrew bolt.
EDGAR M. BICK. Immediate results in pathological fractures.
ALBERT J. SCHEIN. End results of hemiphalangectomy for hallux valgus.

### NEW YORK ORTHOPAEDIC HOSPITAL

Staff—9. Symposium on tuberculosis of joints.
WALKER E. SWIFT. Pathology and diagnosis.
ALAN DEF. SMITH. Treatments, general consideration and reasons for operative treatment.
Demonstration of patients illustrating end results:
WILLIAM H. VON LACKUM. Spine.
HALFORD HALLOCK. Hip, elbow.
GEORGE L. INGE. Knee.
LEONIDAS A. LANTZOUNIS. Ankle.
M. BECKETT HOWORTH. Shoulder.
FREDERICK L. LIEBOLT. Wrist.
GEORGE L. INGE—1:30. Outpatient clinic; general orthopedic conditions.
MALCOLM B. COUTTS—1:30. Club feet.

### RIVERSIDE HOSPITAL

HENRY MILCH—10. Bone complications in advanced pulmonary tuberculosis.

### HOSPITAL FOR RUPTURED AND CRIPPLED

Staff—2. Presentation of orthopedic problems.
RUFUS H. ALLDREDGE. Localized osteitis fibrosa cystica.
RICHMOND STEPHENS. Operative treatment of flat feet.
JOHN R. COBB. Scoliosis.
ERNEST E. MYERS. Low back pain.
T. CAMPBELL THOMPSON. Epiphysiodesis.
PHILIP D. WILSON. Elbow fractures; carpal injuries.
LEWIS C. WAGNER. Tendon transplantation for paralytic wrist drop.
FRANCIS CARR. Fractures of the lower end of the femur.
T. CAMPBELL THOMPSON. Presentation of cases for operation Friday morning.

### UNITED STATES MARINE HOSPITAL

W. G. DORAN—2. Operations.

### VETERANS ADMINISTRATION HOSPITAL

T. F. CARROLL—12:45. Demonstrations: Artificial limb assembly, modified and improved braces, fabricated jackets and supports, orthopedic shoes.
CHARLES F. BLOOM—12:45. Roentgenological equipment, presentation of x-ray material, films and lantern slides of current cases and others of particular interest to the general surgeon and cancer specialist.
C. R. BROOKE—12:45. Physical therapy department, newer modalities of particular interest to the surgeon.

## *Friday*

### GOUVERNEUR HOSPITAL

WALTER D. LUDLUM, JR., JOHN P. STUMP, and staffs—9. Presentation of cases from orthopedic and traumatic services.
CARL A. PETERSON. Fractured femur with sciatic nerve injury.
DR. CLUG. Fractures of the jaw, two cases.
WALTER D. LUDLUM, JR. Selective neurectomy. Incidence of open correction of fresh fractures.
DANTE P. DAPOLONIA and MILES C. KREPELA. Fracture of the humeral epicondyles, open reduction, two cases.
JOHN P. STUMP. Painful knees due to faulty posture, with illustrations. Maintaining reduction of oblique fractures with ambulatory Kirschner wire spreaders.
MILES C. KREPELA. Fracture of the tibial condyles, three cases.
ABRAHAM A. KATZ. Club feet, conservative treatment, two cases.

### HOSPITAL FOR JOINT DISEASES

SAMUEL KLEINBERG, JOSEPH BUCHMAN and staff—9. Dry clinics.
SAMUEL KLEINBERG. Cases of chronic slipped femoral epiphysis treated by open operation; cases of arthroplasty of the hip and knee.
JOSEPH BUCHMAN. Cases of chronic osteomyelitis under maggot treatment; case of Perthes' disease complicating rickets.
JOSEPH BUCHMAN and HERMAN LIEBERMAN. Cases of syphilis of bones and joints.
M. HERZMARK. Case of periosteal fibrosarcoma of the lower end of the femur, six years postoperative, no recurrence, no amputation.
HERMAN LIEBERMAN. Cases of club feet treated by manual correction.
R. R. GOLDENBERG. Case of Perthes' disease complicating a dislocation of the hip.
F. I. SCHWARTZBERG. Cases of osteochondritis of the ischiopubic region.
SAMUEL KLEINBERG, JOSEPH BUCHMAN and staff—9. Operations: Arthroplasty; slipped femoral epiphysis; Perthes' disease; osteomyelitis.
LEO MAYER and staff—2. Operations: Reconstructive tendon surgery.

### LINCOLN HOSPITAL

ARMITAGE WHITMAN—9. Operative and dry clinic.

### NEW YORK ORTHOPAEDIC HOSPITAL

BENJAMIN P. FARRELL and staff—9. Operations.
BENJAMIN P. FARRELL—1:30. Outpatient clinic; general orthopedic conditions.

### HOSPITAL FOR RUPTURED AND CRIPPLED

PHILIP D. WILSON and staff—9. Operations.
Staff—11. Presentation of orthopedic problems.
LEWIS C. WAGNER. High osteotomy for lesions of the hip.
PHILIP D. WILSON. Slipped femoral epiphysis.
T. CAMPBELL THOMPSON. Stabilization of equinus foot.
RAYMOND W. LEWIS and WALTER GRAHAM. Secondary osteo-arthritis following fractures of the ankle.
LEWIS CLARK WAGNER. Plastic operation for recurrent dislocation of the patella.
JOHN R. COBB. Fractures of surgical neck of the humerus.
KRISTIAN D. HANSSON. Paralysis of the serratus magnus.

### SYDENHAM HOSPITAL

HARRY D. SONNENSCHEIN, JOSEPH G. WISHNER, MARK M. YOUMANS, and ADOLPH A. SCHMIER—9. Dry clinic: Fractures of shoulder, wrist and elbow.

# OTOLARYNGOLOGY

## *Monday*

### FLOWER-FIFTH AVENUE HOSPITAL

J. A. W. HETRICK and staff—2. Operations and demonstrations of interesting cases. Mastoid operations.
M. S. LLOYD—2. Bronchoscopic demonstrations.
Motion pictures and exhibits in Trustees' Room.

### HARLEM EYE AND EAR HOSPITAL

CHARLES B. MEDING—9. Tonsil operation.
ALBERT HETHERINGTON and I. HENRY ALEXANDER—2. Demonstrations of cases.

### LUTHERAN HOSPITAL

CHARLES C. FRANCIS and staff—2. Operations.

### MANHATTAN EYE, EAR AND THROAT HOSPITAL

JAMES G. DWYER, C. M. GRIFFITH, and staff—2. Otological operations.
DAVID H. JONES, O. L. MONROE, and staff—2. Rhinolaryngological operations.

### MONTEFIORE HOSPITAL

A. A. SCHWARTZ—2. Dry clinic: Laryngeal tuberculosis.

### MORRISANIA CITY HOSPITAL

G. B. GILMORE and staff—2. Demonstration of interesting cases.

### NEW YORK HOSPITAL

ARTHUR PALMER and staff—2. Operative and dry clinics.
ALFRED F. HOCKER. Upper respiratory carcinoma.
ARTHUR PALMER. Laryngeal stenosis.
E. MILES ATKINSON. Vertigo (aural).
SAMUEL F. KELLEY. Sinusitis in allergic cases.
GERVAIS W. MCAULIFFE. Demonstration of teaching models.

### NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

ARTHUR NILSEN and staff—2. Operations.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

MAX HALLE—1. Lecture.
WILLIAM L. GATEWOOD—2. Operations.

*Tuesday*

BELLEVUE HOSPITAL

J. WINSTON FOWLKES—2. Operations.

BETH ISRAEL HOSPITAL

SAMUEL J. KOPETZKY and staff—2. Operations.

FLOWER-FIFTH AVENUE HOSPITAL

J. A. W. HETRICK and staff—2. Operations and demonstrations of interesting cases. Motion pictures.

HARLEM EYE AND EAR HOSPITAL

ALEXANDER LASZLO—2. Operations and demonstrations of cases.

HARLEM HOSPITAL

HERMAN J. BURMAN and staff—1:30. Endoscopic demonstrations.
HERMAN J. BURMAN, MAX MARSH, MARK GOTTLIEB, and MICHAEL ROSENBLUTH—1:30. Operative and dry clinics.

LENOX HILL HOSPITAL

JOHN D. KERNAN and staff—9. Bronchoscopic clinic.
JOHN D. KERNAN, GIRARD OBERRENDER, and staff—2. Operations.

LINCOLN HOSPITAL

WILLIAM H. HOLDEN, MURRAY BERGER, and HAROLD LIGGETT—2. Operative and dry clinics.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

ROBERT E. BUCKLEY, A. S. WILSON, and staff—9 and 2. Rhinolaryngological clinics.
JOHN R. PAGE, R. H. HUVELL, and staff—9 and 2. Otological clinics.
DAVID H. JONES and JOHN M. LORE—2. Endoscopic demonstration.

METROPOLITAN HOSPITAL

J. A. W. HETRICK and staff—1:30. Tonsil surgery in children.

MOUNT SINAI HOSPITAL

JACOB L. MAYBAUM, WALTER L. HORN, SAMUEL ROSEN, JOSEPH G. DRUSS, HARRY ROSENWASSER, and EUGENE R. SNYDER—10. Operative and dry clinics; intracranial complications of otitic origin; recovered cases of streptococcus meningitis, petrositis; sepsis of otitic origin; value of sulfanilamide in otological conditions; neuro-otological cases; histopathological studies.
RUDOLPH KRAMER and staff—2. Operative and dry clinics.

NEW YORK CITY HOSPITAL

OTTO C. RISCH and staff—2. Operative and dry clinics.
HILTON H. STOTHERS—2. Bronchoscopic clinic.

NEW YORK EYE AND EAR INFIRMARY

J. C. HANLEY—10. Operations and demonstration of interesting cases.
F. C. CARR and W. C. DENISON—2. Operations.

NEW YORK HOSPITAL

Staff—2. Exhibits and demonstrations in outpatient department; pavilion rounds.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

DAVID H. JONES—9. Bronchoscopy, lecture and demonstration.
JULIUS I. KLEPPER—11. Cadaver demonstration.
RALPH ALMOUR—1. Cadaver demonstration.
SAMUEL J. KOPETZKY—2. Cadaver demonstration.

PRESBYTERIAN HOSPITAL

JAMES W. BABCOCK—2. Operations.

RIVERSIDE HOSPITAL

GEORGE D. WOLF and DAVID I. FRANK—10. Operations: Direct laryngoscopy and cauterization of larynx. Dry clinic: Tuberculous laryngitis in various stages.

ROOSEVELT HOSPITAL

CHARLES N. HARPER and staff—2. Bronchoscopic clinic.

ST. FRANCIS' HOSPITAL

HENRY J. DILLEMUTH and staff—2. Operations and demonstrations of cases.

ST. VINCENT'S HOSPITAL

JOHN M. LORE and staff—2. Presentation of cases: Stripping of vocal cords; laryngofissure for carcinoma; laryngectomy; laryngeal stenosis; subglottic tumor in a child; cases for diagnosis. Lantern slide talks: Technique of stripping of vocal cords; Lore modification of laryngofissure operation; operative procedure for relief of double abductor paralysis.
ANTHONY ROTTINO—2. Pathological demonstration.

*Thursday*

BELLEVUE HOSPITAL

J. WINSTON FOWLKES and staff—2. Dry clinic: Postoperative petrositis; motion pictures of radical frontal sinus operation.

FLOWER-FIFTH AVENUE HOSPITAL

J. A. W. HETRICK and staff—2. Demonstrations of interesting cases.

HARLEM EYE AND EAR HOSPITAL

ALEXANDER LASZLO—2. Mastoid operations.

HOSPITAL FOR JOINT DISEASES

JULIUS A. HAIMAN and staff—9. Dry clinics.
JULIUS A. HAIMAN and staff—2. Operations.

LENOX HILL HOSPITAL

GIRARD OBERRENDER and staff—9. Bronchoscopic clinic.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

JOSEPH D. KELLY, C. W. DEPPING, and staff—9 and 2. Otological clinics.
FRANCIS W. WHITE, J. D. WHITHAM, and staff—9 and 2. Rhinolaryngological clinics.

MORRISANIA CITY HOSPITAL

CLARENCE H. SMITH—2. Operations: Mastoid.

NEW YORK CITY HOSPITAL

HAROLD B. JUDD—2. Operative and dry clinics.

NEW YORK EYE AND EAR INFIRMARY

EDGAR BURCHELL—10. Dry clinic: Surgical anatomy of the temporal bone and its variations.
J. M. SMITH—10. Operations.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

ARTHUR NILSEN and staff—2. Operations.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

HENRY B. ORTON—9. Cadaver demonstration.
J. EASTMAN SHEEHAN—11. Lecture on otolaryngology and plastic surgery.
W. WALLACE MORRISON—1. Lecture.
LEE M. HURD—2. Operations.
H. G. BULLWINKEL—3. Cadaver demonstration, bronchoscopy.

PRESBYTERIAN HOSPITAL

GEORGE R. BRIGHTON—2. Bronchoscopic clinic: Presentation of cases; demonstration of technique of bronchospirometry.

ROOSEVELT HOSPITAL

CHARLES N. HARPER and R. C. GROVE—2. Dry clinic.

ST. LUKE'S HOSPITAL

WESLEY C. BOWERS and staff—2. Operative and dry clinics: Meningeal infections of otologic origin; sinus infections and their treatment.

UNITED STATES MARINE HOSPITAL

W. P. GRIFFEY, A. J. HUEY, and staffs—2. Operations.

*Friday*

FLOWER-FIFTH AVENUE HOSPITAL

J. A. W. HETRICK and staff—2. Operations and demonstrations of interesting cases; motion pictures.

HARLEM EYE AND EAR HOSPITAL

JOHN J. LEVBARG—9. Voice and speech clinic.
Staff—2. Operations and demonstrations of cases.

HARLEM HOSPITAL

HERMAN J. BURMAN, NATHANIEL HALPERN, and CHARLES HARRIS—9. Tonsil clinic: Snaretone and dissection operations.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

MARVIN F. JONES, D. S. CUNNING, T. G. TICKLE, and staff—9 and 2. Otological clinics.
E. ROSS FAULKNER, WESLEY M. HUNT, and staff—9 and 2. Rhinolaryngological clinics.
DAVID H. JONES and JOHN M. LORE—2. Endoscopic demonstration.

METROPOLITAN HOSPITAL

J. A. W. HETRICK and Staff—1:30. Operative and dry clinics.

MORRISANIA CITY HOSPITAL

G. B. GILMORE, HENRY J. DILLEMUTH, and JOSEPH LASALA—2. Bronchoscopic clinic.

MOUNT SINAI HOSPITAL

RUDOLPH KRAMER and Staff—2. Operative and dry clinics.

NEW YORK EYE AND EAR INFIRMARY

T. L. SAUNDERS—10. Demonstration of interesting ear and sinus cases; operations.
STUART L. CRAIG—2. Operations.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

PAUL S. SEAGER—2. Laryngeal surgery, cadaver.
CHARLES M. GRIFFITH—2. Sinus surgery, cadaver.
JAMES O. MACDONALD—2. Mastoid surgery, cadaver.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

M. COLEMAN HARRIS—9. Allergy in otolaryngology.
NATHAN SETTEL—10. Lecture.
THOMAS G. TICKLE—11. Cadaver demonstration of facial palsy.
MALCOLM W. CARR—1. Oral surgery in otolaryngology.
CHARLES J. IMPERATORI—2. Operations.
M. H. KAIDEN—3. Lecture.

## OPHTHALMOLOGY

*Monday*

HARLEM EYE AND EAR HOSPITAL

HARVEY LYON—2. Refraction clinic.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

FRANK C. KEIL and staff—2. Operations.

MORRISANIA CITY HOSPITAL

JOSEPH S. HORY and staff—2. Operations for cataract, glaucoma and squint; interesting eye conditions in relation to general medicine.

NEW YORK EYE AND EAR INFIRMARY

CLYDE E. MCDANNALD and staff—2. Operations: Cataracts; muscle; ptosis.
TRUMAN L. BOYES—4. Surgical treatment of ocular muscle anomalies with demonstrations of cases.

NEW YORK HOSPITAL

Staff—2. Dry clinics.
BERNARD SAMUELS. Intra-ocular tumors, lecture illustrated with anatomical preparations. Demonstration of anatomical specimens and teaching models.
MILTON L. BERLINER. Slit lamp demonstration.
N. BLAIR SULOUFF. Practical points in refraction.

NEW YORK CITY HOSPITAL

RAYMOND E. MEEK and FRANK C. KEIL—2. Operative and dry clinics.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

ERVIN TOROK—3. Operations.

RIVERSIDE HOSPITAL

CLIFFORD W. ELLISON—3. Eye conditions in tuberculous patients.

*Tuesday*

BELLEVUE HOSPITAL

WEBB W. WEEKS and staff—2. Operations for cataract, glaucoma and squint.

HARLEM EYE AND EAR HOSPITAL

CHARLES B. MEDING and ARCHIE OBERDORFER—2. Operations and demonstrations of cases.

HARLEM HOSPITAL

PAUL A. COLLINS—2. Fundus operations and ward rounds.

KNAPP MEMORIAL EYE HOSPITAL

ARNOLD KNAPP and staff—9. Operative and dry clinics.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

PLINEUS H. MONTALVAN—9. Follow-up clinic on glaucoma.
McCLELLAND SHELLMAN—9. Fundus clinic.
H. ROBERTSON SKEEL and staff—2. Operations.

METROPOLITAN HOSPITAL

A. L. CHAMBERS and staff—1:30. Fundus clinic, external diseases of the eye.

MONTEFIORE HOSPITAL

SIGMUND A. AGATSTON—9. Dry clinic.

NEW YORK EYE AND EAR INFIRMARY

E. B. BURCHELL—9:30. The laboratory and its aid to ophthalmology.
IRVING SCHWARTS—10:30. X-ray of accessory sinuses and skull; fundamentals.
WILLIS S. KNIGHTON—11:30. Contact lenses.
SAMUEL P. OAST and staff—2. Operations: Cataracts; modified LaGrange; general.
BERNARD SAMUELS and staff—2. Operative and dry clinics.

NEW YORK HOSPITAL

Staff—2. Exhibit of rare old English, American, German and French books on ophthalmology; interesting clinical cases.

NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL

CLYDE E. McDANNALD—3. Operations.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

MARTIN COHEN—2. Dry clinics.

PRESBYTERIAN HOSPITAL
(Institute of Ophthalmology)

JOHN M. WHEELER and staff—9:30. Operative and dry clinics.

UNITED STATES MARINE HOSPITAL

W. P. GRIFFEY and RUDOLPH AEBLI—2. Operative and dry clinic.

*Thursday*

BETH ISRAEL HOSPITAL

WEBB W. WEEKS—2:30. Operations.

COLUMBUS HOSPITAL

GIROLAMO BONACCOLTO—9:30. Exhibit of slit lamp cases and operations.

HARLEM EYE AND EAR HOSPITAL

JOHN J. DECKER, MAX GOLDSCHMIDT, and LOUIS SCHWARTS—2. Operations and demonstrations of cases.

HARLEM HOSPITAL

HENRY MINSKY—2. Operative and dry clinics; Lid injuries.

LENOX HILL HOSPITAL

E. F. KRUG, J. J. REID, JR. and staff—2. Operations.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

PLINEUS H. MONTALVAN—9. Demonstration of contact glasses and telescopic lenses.
ANDREW A. EGGSTON and JOSEPH LAVAL—9. Demonstrations of eye pathology.
NORTON DeL. FLETCHER and staff—2. Operations.

MOUNT SINAI HOSPITAL

KAUFMAN SCHLIVEK and staff—9. Cataract extraction, trephine, LaGrange, Safar for detachment, ptosis-advancement of levator.

NEW YORK EYE AND EAR INFIRMARY

CONRAD BERENS and staff—9. Operations: Iridocorneosclerectomy; retroplacements; Tenon's capsule transplant; iridocapsulotomy; general operations.
WEBB W. WEEKS and staff—2. Operations: Intracapsular cataracts; modified LaGrange; muscle operations.
WENDELL L. HUGHES—2. Plastic; corneal transplant.

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

MARTIN COHEN—2. Dry clinic.

PRESBYTERIAN HOSPITAL
(Institute of Ophthalmology)

JOHN A. DUNNINGTON, THOMAS H. JOHNSON, DANIEL B. KIRBY, and ALGERNON B. REESE—9:30. Operative and dry clinics.

*Friday*

BELLEVUE HOSPITAL

WEBB W. WEEKS and staff—2. Demonstrations of postoperative cases, illustrated with results from operations for cataract, glaucoma and plastic surgery of the eye and orbit.

HARLEM EYE AND EAR HOSPITAL

CHARLES B. MEDING and staff—2. Operations and demonstrations of cases.

HOSPITAL FOR JOINT DISEASES

DAVID WEXLER and staff—2. Dry clinic.
MAURICE POMERANZ and HENRY K. TAYLOR—2. Planigraphy in orthopedic surgery.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

R. TOWNLEY PATON—9. Demonstration of cornea transplanting and ophthalmic photography.
GIROLAMO BONACCOLTO—9. Slit lamp demonstration.
DAVID H. WEBSTER and staff—2. Operations.

PRESBYTERIAN HOSPITAL
(Institute of Ophthalmology)

JOHN A. DUNNINGTON, THOMAS H. JOHNSON, DANIEL B. KIRBY, and ALGERNON B. REESE—9:30. Operative and dry clinics.

ST. LUKE'S HOSPITAL

W. GUERNSEY FREY, JR. and staff—2. Operative and dry clinics.
H. T. SMITH. The Laurence-Moon-Biedl syndrome.
WALTER HIPP. Treatment of conjunctivitis with bacteriophage.

## BROOKLYN-LONG ISLAND DAY—WEDNESDAY

### Operative and Dry Clinics—9–12:30

### BROOKLYN CANCER INSTITUTE

#### Operative Clinics—9

J. J. Gainey and George Reitz. Radical mastectomy, two cases.
J. McGoldrick. Carcinoma of the cervix.
I. Sirus. One-stage Miles resection for carcinoma of rectosigmoid.
G. Robillard. Surgery of the colon.
E. K. Morgan. Ureterostomy for carcinoma of bladder.

#### Dry Clinics—9

W. E. Howes. Pre-operative radiation of the breast.
S. G. Schenck. Bronchial carcinoma and review of six autopsied cases.
H. Charache. Neurogenic sarcoma. Eighteen cases with statistical study and lantern slides.
J. Schmidt. Carcinoma of the larynx; presentation of operated and radiated cases, wax model demonstration.
H. S. Rasi. Oral surgery in malignancy, with wax model exhibit.
S. Wolfe. Carcinoma of the vulva, case presentations.
L. S. Drexler. Carcinoma of the genito-urinary tract, lantern slide demonstration.
H. Bolker and H. Koppelman. The effect of radiation on mammary tissue and breast cancer.
H. I. Teperson. X-ray treatment of giant cell tumors. Demonstration of cases.

### BROOKLYN HOSPITAL

Ernest K. Tanner and staff—9. General surgical operations.
Ernest K. Tanner, D. D. Davis, and W. H. Field. Lobectomy for bronchiectasis; transposition of viscera.

#### Clinical Presentations

Ernest K. Tanner. Review of one year's anesthesia. Review of empyema for eighteen years.
D. D. Davis. Massive drip blood transfusion from blood bank.
W. H. Field. Study of the results of gall bladder surgery, two five-year periods.
R. B. Miles. Comparative study of drainage versus non-drainage in acute appendicitis.
A. L. Smith. Analysis of surgical mortality.
S. A. Winning. End-results of sympathectomy in Raynaud's disease.
E. Jefferson Browder—9. Neurosurgical operations. Surgical treatment of epilepsy. Demonstration of bipolar cortical stimulation. Posterior fossa approach for major trigeminal neuralgia.
Nathaniel P. Rathbun and staff—9. Genito-urinary surgical, operative and dry clinics.
N. P. Rathbun, H. Fisher and W. F. McKenna. Tidal drainage of the bladder with control of time, quantity, and pressure factors.
H. Wehrbein. Rupture of renal cyst with extravasation of urine forming extrarenal urinary cyst.
F. C. Hamm. Motion pictures of genito-urinary surgery in children.
F. C. Bond. Ureteropelvic obstructions. Series of operated cases and x-rays.
William Sidney Smith and staff—9. Gynecological and obstetrical, operative and dry clinics.
W. S. Smith, Eliot Bishop, John Casagrande, and George G. Cochran. Dry clinic: Outline of combined medical-obstetrical clinic for care of cardiac patients. Experiences with vaginal antisepsis. Survey of the obstetrical service. Diagnosis and treatment of hydatidiform mole.
Donald E. McKenna and staff—9. Orthopedic surgery.
Donald E. McKenna. Operations: Arthrotomy of the knee joint.
Henry Lange. Xanthoma of knee joint.
L. Gaston Papae. Disease of the spine with symptoms simulating lesions of the urinary tract.
A. W. Martin Marino and staff—9. Rectal surgery. Abdominoperineal resection for cancer of rectum. Other proctological operations. Demonstration of patients who have undergone different types of operations for cancer of rectum. Visit to rectal clinic.

### CUMBERLAND HOSPITAL

#### Operative Clinics

Merrill N. Foote and staff—9. General surgical operations.
Carlton Campbell. Physiological gastric resection.
Rudolph Goldberg. Thyroidectomy.
Howard Blair. Cholecystectomy. The use of stainless steel wire in abdominal closures.
John Timm and staff—9. Operations. Splenectomy for Banti's disease.
Stanley D. Banks. Radical mastectomy for carcinoma of breast.
Eugene F. Vitagliano. Hysterectomy for fibroid.
Herbert T. Wikle and staff—9. Operations. Bone dust hernioplasty.
Henry Wagener. Gastric resection.
Howard T. Langworthy and staff—9. Genito-urinary surgical operations.
Leo S. Drexler. Prostatectomy. Nephrectomy.
Frank H. Lasher and staff—9. Otolaryngological operations.
Abraham G. Silver, Louis R. Usen, Bernard N. Gottlieb, Samuel Kaplan. Bronchoscopy. Lateral sinus thrombosis. Nose and throat operations.

#### Dry Clinics

William C. Meagher and staff—9. Obstetrical dry clinic.
William C. Meagher. Cesarean sections at Cumberland Hospital.
Samuel Lubin. Results of attempted induction of labor with estrin.
Alexander Dunbar. Operative deliveries at Cumberland Hospital.
Staff—10. General surgery, dry clinic.
Harry Martz. Analysis of 50 consecutive ruptured gastric ulcers, lantern slides.
Jack Romascan. Familial neurotrophic osseous atrophy, demonstration of cases.
Carlton Campbell. Presentation of cases of mammary tuberculosis.
Saul F. Livingston. Granuloma inguinale, end-results of cases treated locally.

MERRILL N. FOOTE and SILIK H. POLAYES. Acute appendicitis, B. welchii blood stream infection, extrusion of entire spleen, cure, presentation of patient.

## JEWISH HOSPITAL

WILLIAM LINDER and staff—9. General surgical operations; resection for carcinoma of the stomach.
HENRY LOURIA and staff—9. General surgical operations.
DAVID FARBER. Thyroidectomy.
FRANK TELLER. Problem of recurrent hyperthyroidism.
LOUIS BERGER and staff—9. General surgical operations and dry clinics.
DAVID TEPLITSKY. Colonic resection for carcinoma.
RALPH WOLFE. Organization of the gastro-intestinal surgical division.
MILTON J. RADER. Dry clinic: Acute appendicitis, twenty-year study at Jewish Hospital.
ROBERT E. ROTHENBERG. Dry clinic: Retroperitoneal cyst.
PAUL ASCHNER and staff—9. Genito-urinary surgical operations and dry clinics.
ABRAHAM PROGEBIN. Nephrectomy for tuberculous kidney.
SAUL PARNASS. Second stage prostatectomy.
LOUIS T. MORSE. Urological approach to treatment of essential hypertension.
PAUL ASCHNER, DAVID M. GRAYZEL, MAX LEDERER, MILTON G. WASCH. Dry clinic: Exhibit of tumors of kidney, ureter, bladder, and prostate; classification, diagnosis, pathology, treatment.
LEO M. DAVIDOFF and ISADORE M. TARLOV—9. Neurosurgical operations. Right transfrontal craniotomy for suprasellar meningioma. Demonstration of electroencephalography.
E. LEO BERGER and staff—9. Otolaryngological operations.
PHILIP LEIBOWITZ, CARL KAPLAN, CHARLES BREITMAN, BERNARD WELT. Radical mastoidectomy. Demonstration of bronchoscope.
LEO S. SCHWARTZ and staff—9. Gynecological and obstetrical operations.
SAMUEL SCHENCK, SAMUEL A. WOLFE, EMANUEL V. LITTAUER. Vaginal plastic operation. Hysterectomy.

## KINGS COUNTY HOSPITAL

### Operative and Dry Clinics—9:30

EDWIN FISKE. Thyroid clinic.
ROBERT F. BARBER. Operation, colon surgery.
EDWARD P. DUNN. Operations, general surgery.
CHARLES B. JONES. Operations, general surgery.
JOSEPH TENOPYR. Fracture demonstration, ward walk.
JOSEPH RAPHAEL. Fracture demonstration, operation.
H. WRIGHT BENOIT. Thyroid operation.
WILLIAM ENNIS. Open reduction of fractures.
NICHOLAS RYAN. Gastric ulcers, statistical study.
OTTOKAR TENOPYR. Fracture demonstration.
WALTER COAKLEY. Plastic surgery, operations, two hours.
CHARLES S. COCHRANE. Ward walk, display of x-ray pictures of kidney, ureter, and bladder.
MATTHEW GOLDEN. Operative and dry clinic.
WALTER MOEHLE. Demonstration of ophthalmic operations.
J. B. L'EPISCOPO. Demonstration of bone block operation for painful hip.
RALPH GARLICK. Plastic operation for prolapse.
JOSEPH MCGOLDRICK. Operation, hysterectomy.
CHARLES MUELLER. Gynecological operations.
HENRY GOUBEAUD. Statistical study of placenta previa.
CHARLES GORDON. Gynecological operation.
MORRIS GLASS. Ward demonstration, cardiac conditions in pregnancy, toxemia in pregnancy.
EDWIN GRACE. Pectoral transplant for coronary occlusion.

## LONG ISLAND COLLEGE HOSPITAL

### Operative Clinics—9

EMIL GOETSCH. Thyroidectomy for exophthalmic goiter.
ALFRED C. BECK. Cesarean section.
FEDOR SENGER and staff. Tumors of the kidney.
RALPH HARLOE. Empyema. Operation. Closed treatment of all types.
WILLIAM A. JEWETT. Fothergill operation.
HARVEY B. MATTHEWS. Vaginal plastic under local anesthesia.
GEORGE W. PHELAN. Tumors of the round ligament.
MERVYN V. ARMSTRONG. Episiotomy and repair under local anesthesia.
GEORGE HORTON. Nephrectomy.

### Dry Clinics—10:30

S. POTTER BARTLEY. Fractures of the foot, clinical and economic aspect, lantern slides.
HERBERT C. FETT. Arthrotomy of the knee joint, report of experiences.
ROBERT F. BARBER. Malignant tumors of peripheral nerves, cases and specimens.
SAMUEL A. WOLFE. Demonstration of ovarian tumors.

## METHODIST EPISCOPAL HOSPITAL

HAROLD K. BELL, HENRY F. GRAHAM, PIERRE A. RENAUD, SEYMOUR G. CLARK, JOHN A. TIMM, and staffs—9. General surgical operations.

### Dry Clinics

S. G. CLARK. Vascular tumors of the liver.
HAROLD K. BELL. Intussusception with Meckel's diverticulum.
MILTON E. HOEFLE. Use of long intestinal tube for relief of intestinal obstruction.
OSCAR P. SCHOENEMANN. Planography in tuberculosis.
HAROLD E. RHAME. Elliott treatment for pelvic inflammations.
C. DOUGLASS SAWYER. The peritoneoscope.
J. H. BLISS. Primary carcinoma in a branchial cyst.
KENNETH H. MACGREGOR. Surgical indications for blood transfusions.
H. RUSSELL MEYERS—9. Neurosurgical dry clinic: Chronic subdural hematoma in infancy.
DONALD E. MCKENNA, HENRY F. GRAHAM, HAROLD K. BELL, JOHN A. TIMM and staffs—11. Fracture clinic.
JOHN A. TIMM. Use of Soutter apparatus for reduction of fractures of both bones of forearm.
HENRY F. GRAHAM. An efficient method for reduction and retention of Colles' fracture.
HENRY P. LANGE. Fracture of radius and ulna, lower fifth, in adolescence.
HOWARD T. LANGWORTHY and ROSARIA P. MULE—9. Genito-urinary surgery, urological exhibit.
ROSARIA P. MULE—11:30. Medical aspects of prostatism.
CHARLES L. STONE, CHARLES A. ANDERSON, EINAR A. SUNDE, and staffs—11. Otolaryngological operations and dry clinics.
CHARLES L. STONE and CHARLES A. ANDERSON. Dry clinic: Ligation of the common carotid artery for hemorrhage.
JOHN F. FORD—9. Plastic surgery, dry clinic: The cosmetic aspect of rhinoplastic surgery.

O. Paul Humpstone, Ralph M. Beach, Harvey B. Matthews, Harry W. Mayes, George H. Davis and staffs—9. Operations and ward rounds in obstetrics.

## ST. JOHN'S HOSPITAL

### Operative and Dry Clinics—9

John E. Jennings. Radical mastectomy with ligation of axillary vein. Treatment of inoperable breast cancer.
Frank Sammis. Grill graft plastic.
Stanley B. Thomas and George R. Marsh. Gastrointestinal surgery. Cancer of colon-megacolon, review of cases.
Augustus Harris. Surgical attack on anomalies of kidney and ureter, cases and results reviewed.
S. Lloyd Fisher. Surgery of the female pelvis.
James L. Cobb and George B. Reitz. Acute and traumatic surgery, operations and demonstrations.
L. Albert Thunig. Anomalies of the umbilicus.

### Co-ordinated Medical and Obstetrical Demonstrations

Cameron Duncan. Handling of labor in tubercular cases, and postpartum care.
Lowell B. Eckerson. Back pain associated with gastrointestinal lesions.
Charles E. Hamilton. Collapse therapy in tuberculosis of the lung.
Carl Greene. The acute gall bladder.
Paul Parrish. Pyloric stenosis in infants.
Fred Maisel. Surgical photography.
John B. Knapp and William Richard Cashion. Roentgenography and x-ray therapy.
Leonid Watter. Anesthesia procedures.

## ST. MARY'S HOSPITAL

William Pascual, Thomas M. Brennan, Peter Dulligan and staffs—9. General surgery, operative and dry clinics.
Sanford Shumway. Selected fracture problems.
Daniel Welsh. Acute intestinal obstruction due to gall stone occlusion.
Hugh Murphy. Mecholyl iontophoresis in the treatment of thrombophlebitis.
Peter Dulligan. Management of acute intussusception.
Thomas Brennan. Cases illustrative of retroperitoneal hemorrhage and other types of abdominal trauma.
William Pascual. Atypical appendicitis, difficulties in recognition and management.
Joseph Rizzo. Appendicitis in pregnancy.
Hugh Murphy and V. Tesoriero. Vascular clinic, demonstration of cases and methods of treatment.
Andrew J. McGowan, Frank C. Hamm and staffs—9. Genito-urinary surgery, operative and dry clinic.
Frank C. Hamm. Transurethral prostatectomy.
Andrew J. McGowan. Pediatric urology.
George Price and William Moitrier, Jr.—9. Pathological demonstration, skin tumors, ovarian tumors in their relationship to sex.
John Shields and staff—9. Proctological dry clinic.
Herbert C. Fett and staff—9. Orthopedic surgery, selected problems.
Albert Keenan, John Auwerda, and staffs—9. Otolaryngological surgery.
E. A. Keyes, H. Goubeaud, C. Loughran, R. Wilson, and staffs—9. Gynecological and obstetrical operations. Vaginal hysterectomy (local), anterior and posterior colporrhaphy (local), panhysterectomy (spinal), laparotomy for ovarian cyst.

### Roentgenological Department—9

Francis Currin. Exhibits.
Paul Raia. Cholangiography in biliary tract disease.
Francis Currin. Radiation in postoperative parotitis.

### Dry Clinic

H. Goubeaud and staff—9. Forceps application (mannikin), Kielland, Pieper, axis traction. Resuscitation of newborn, cadaver and motion pictures. Graphs and charts: dehydration, mortality, morbidity, cesarean sections, repeat sections, x-ray in placenta previa. Blood bank in obstetrics.
M. Murphy. Eight cesarean sections, ectopic pregnancy.
F. Mitchell. Double pregnancy, double uterus, abortion at fourth month, first uterus; full term cesarean section, living baby, second uterus.
Charles Loughran. Hydatidiform mole, cesarean section at term, living baby.
M. Murphy. Two cases abdominal hemorrhage, tubal in origin, not ectopic.
J. Mastrota. Antepartum diagnosis, triple pregnancy, with x-ray.
H. Joyce. Electric cervix, advantages and disadvantages.
M. Abbene. Hydatidiform mole, analysis of nineteen cases.

## UNITED STATES NAVAL HOSPITAL

### Operative Clinics—9

Charles H. Savage. Appendectomy, spinal anesthesia.
James J. O'Connor. Repair of inguinal hernia, local anesthesia.
Courtney G. Clegg. Rectal clinic.

### Dry Clinics—10

Louis E. Gilje. Fracture clinic.
Calvin B. Galloway. Cancer clinic.

## BROOKLYN EYE AND EAR HOSPITAL

### Ophthalmology

*Morning Session—9:30. Dry Clinics*

E. Clifford Place. Keratoconus treated with contact lenses. Sarcoma of the choroid.
Charles A. Hargitt. Hereditary juvenile glaucoma, two families.
Ralph I. Lloyd. Birth injuries of the cornea and allied conditions.
Walter V. Moore. Scleromalacia perforans, report of four cases.
William F. C. Steinbugler. Ophthalmomyiasis anterior.
Mortimer A. Lasky. Krukenberg's spindle.
Daniel Kravitz. Hodgkin's disease of the lid.
Irving Jacobs. Sperophakia, ectopia lentis with lens extraction O.U.
John N. Evans. Preparation for operation and aftercare of glaucoma cases.

### Motion Picture Demonstrations

P. Chalmers Jameson. Jameson recession.
Walter Moehle. Cataract extraction, a new technique.

*Afternoon Session—2:30. Operative Clinics and Symposium*

John H. Ohly, David T. Bishop, and staffs. Symposium on retinal detachment.
P. Chalmers Jameson and E. Clifford Place. Operative clinics.

CHARLES A. HARGITT, JOHN N. EVANS, WALTER V. MOORE and staffs. Demonstration of patients.

OTOLARYNGOLOGY

*Morning Session—9. Operative and Dry Clinics*

HARRY PATRIE. Osteomyelitis of temporal bone. Case report and patient.
ALEXANDER HOWE. Frequency of dehydration after mastoidectomy.
SAMUEL ZWERLING. Chilliness—a symptom of nasal obstruction.
ROBERT L. MOORHEAD. Petrous pyramid suppuration, cases, demonstration on cadaver.
JOHN AUWERDA. Bronchoscopy, interesting cases.
JOHN P. BAKER. Cancer of the larynx, laryngectomy and laryngofissure patients.
WILLIAM HERBERT. Ultra short wave therapy.
EDWARD E. WOODLAND. Evolution of ear, nose, and throat specialty in the Medical Corps U. S. Navy—25 years.
EDWARD A. BERGER. Demonstration by specimens of carotid approach to petrous pyramid.
Operative clinics by staff. Operations and operators to be announced later.

## SYMPOSIA—2:00–4:00

### GENERAL SURGERY

*Biliary Surgery—Jewish Hospital—Moderator:* LOUIS BERGER

LOUIS J. MORSE. Clinical analysis of gall bladder disease at the Jewish Hospital for the past ten years.
THEODORE BARNETT. Study of intraductal and sphincter pressure in common duct drainage cases.
BERT B. HERSHENSHON. Anesthesia—its relation to surgery of the gall bladder.
DANIEL A. MCATEER. Physiology of the liver and biliary tract.
RUSSELL S. FOWLER. Why to operate, when, and what operation to do in acute cholecystitis.
STUART A. WINNING. Pre-operative and postoperative care in biliary surgery.

*Surgery of the Thyroid Gland—Long Island College Hospital—Moderator:* EMIL GOETSCH

EMIL GOETSCH. Operative and postoperative reactions in thyroidectomy for hyperthyroidism.
ARTHUR GOETSCH. Hyperthyroidism in children.
BENJAMIN CISSEL. Pathological conditions of the thyroid gland.
HAROLD K. BELL. Care of thyroid cases on a general surgical service.
EDWIN H. FISKE. Amount of thyroid gland to be removed in mild hyperthyroid cases.
HARRY FELDMAN. A safe and standardized thyroidectomy technique, colored motion pictures.
A. STORRS WARINNER. Tuberculosis of the thyroid gland.

*Gastric Surgery—Methodist Episcopal Hospital—Moderator:* HENRY GRAHAM

GEORGE W. CRAMP. X-ray diagnosis of gastric lesions.
ALBERT F. R. ANDRESEN. The 1938 ulcer diet, physiological basis, Meulengracht diet.
W. WRIGHT BENOIT. Pyloric obstruction in infancy.
JOHN E. HAMMETT. Surgical treatment of ulcer.
WILLIAM LINDER. Surgical treatment of cancer of the stomach.
PIERRE A. RENAUD. Important points in the technique of gastric operations.
J. HERBERT BLISS. Gastrojejunal ulceration.
WILLIAM H. FIELD. Causes of failure after gastric operations.

*Thoracic Surgery—Kings County Hospital—Moderator:* EDWIN J. GRACE

EDWIN J. GRACE. Address of welcome.
IRWIN E. SIRIS. Lobectomy for bronchiectasis.
WILLIAM H. FIELD. Review of postoperative specimens of bronchiectasis.
WILLIAM E. HOWES. Exhibit of bronchial carcinoma with review of autopsied specimens.
ARTHUR S. W. TOUROFF. Acute abscess of the lung.
EDWIN J. GRACE and CHALMER D. DIXON. Phrenic nerve operations for pulmonary tuberculosis, end-results of 300 cases.
RALPH F. HARLOE. The problem of empyema in pulmonary tuberculosis.

*Lesions of the Breast—St. John's Hospital—Moderator:* JOHN E. JENNINGS

JOSEPH TENOPYR and IRVING SILVERMAN. Diagnosis of tumors of the breast, new biopsy punch.
L. ALBERT THUNIG. Frozen section technique.
WILLIAM E. HOWES. Pre-operative radiation of the breast.
HAROLD KOPPELMAN and HERMAN BOLKER. Effect of radiation on mammary tissue and breast carcinoma.

*Surgery of the Rectum and Colon—Brooklyn Hospital—Moderator:* ERNEST K. TANNER

J. HERBERT BLISS. Cancer of the rectum, surgical methods of approach with special consideration of one-stage resection.
GEORGE WEBB. Use of Abbott tube in intestinal obstruction.
JAMES WATT. Acute obstruction of the large intestine.
A. W. MARTIN MARINO. Review of 114 cases of carcinoma of the rectum and rectosigmoid.
ARTHUR HOLZMAN. Surgery of the ascending colon.
BENJAMIN SEAMAN. The Devine operation in colon surgery.
BERNARD S. PUPEK. Treatment of carcinoma of the colon.

*Surgery of Peripheral Vascular Diseases—St. Mary's Hospital—Moderator:* ROBERT F. BARBER

HAROLD RABINOWITZ. Treatment of thrombo-angiitis obliterans based upon new concepts of the pathogenesis and pathological physiology of the disease; lantern slides.
HUGH L. MURPHY. Experience in treating peripheral vascular disease with pavaex, evaluation of results in over 125 cases. New method in treating thrombophlebitis with mecholyl iontophoresis.
FRANK N. DEALY. Surgery of peripheral vascular diseases.
ROBERT F. BARBER. Experience with aneurysm.
HERBERT T. WIKLE. Allergy simulating peripheral vascular disease.

*Acute Osteomyelitis—St. Mary's Hospital—Moderator:* WILLIAM V. PASCUAL

DANIEL E. WELCH. Multiple lesions of acute osteomyelitis.
JOHN M. SCANNELL. Modern treatment of acute osteomyelitis in children.

AINSWORTH L. SMITH. Conservative operating in acute osteomyelitis.
SEYMOUR G. CLARK. Etiology of acute osteomyelitis.
S. LLOYD FISHER. Diagnosis in acute osteomyelitis.
DANIEL A. MCATEER. Pathology of acute osteomyelitis.

## OBSTETRICS AND GYNECOLOGY

*Hemorrhage Associated with Pregnancy — Methodist Episcopal Hospital—Moderator:* O. PAUL HUMPSTONE

ONSLOW A. GORDON. Abortion.
ELIOT BISHOP. Unusual antepartum lesions.
RALPH M. BEACH. Placenta previa.
G. HAMILTON DAVIS. Ablatio placentæ.
HENRY J. GOUBEAUD. Ruptured uterus.
HARRY W. MAYES. The postpartum period.
HARVEY B. MATTHEWS. The puerperium.

*Maternal Welfare and Toxemias of Pregnancy—Long Island College Hospital—Moderator:* ALFRED C. BECK.

There will be a one hour demonstration under the Chairmanship of Dr. Charles Gordon of the actual conduct of the Committee on Maternal Welfare of the Medical Society of the County of Kings. This Committee has for its purpose the improvement of obstetrics by an analysis of the causes of all maternal deaths, the assignment of the responsibility for the same, and suggested remedies.

Toxemias of Pregnancy

MERVYN V. ARMSTRONG. The management of hyperemesis gravidarum.
FRANK P. LIGHT. Nonconvulsive late toxemia.
ALFRED C. BECK. Convulsive late toxemia.

## FRACTURES AND TRAUMATIC SURGERY

*Cumberland Hospital—Moderator:* FUAD I. SHATARA

JOSEPH RAPHAEL. Observations on local anesthesia in the reduction of fractures, report of 100 cases.
HAROLD DRAFFEN. Treatment of compound fractures.
ARCHIE M. BAKER. Fracture clinic in a small hospital.
J. EARL MILES. Pinning of fractures about the elbow joint.
GEORGE B. REITZ. Transportation of head injuries. Madelung's deformity, value in diagnosis of dislocation of the wrist.
JOSEPH I. ANTON. Treatment of nerve injuries.
LEO FASKE. Trimalleolar fracture.
S. POTTER BARTLEY. Management of fractures of the os calcis.

## GENITO-URINARY SURGERY

*Treatment of Malignant Diseases of the Genito-Urinary Tract—The Brooklyn Hospital—Moderator:* NATHANIEL P. RATHBUN.

HENRY H. MORTON. Address of welcome.
FEDOR L. SENGER. The kidney and ureter.
HEINRICH L. WEHRBEIN. The adrenal.
PAUL WILLIAM ASCHNER. The bladder—clinical application of pathologic data.
EDWIN KING MORGAN. The bladder—endovesical treatment.
AUGUSTUS HARRIS. The bladder—open surgical treatment.
HOWARD LANGWORTHY. The penis.
J. STURDIVANT READ. The prostate.
LT. C. B. GALLOWAY. Present day management of teratoma of the testicle.

## SURGERY OF THE BONES AND JOINTS

*Kings County Hospital—Moderator:* JOSEPH B. L'EPISCOPO

CHARLES D. NAPIER. Vertebral epiphysitis in adolescence.
HERBERT C. FETT. Fractures of the forearm in children.
OTHO C. HUDSON. Fracture of the astragalus.
BENJAMIN KOVEN. Osteomyelitis of the spine treated by graft fusion; lantern slides.
JOSEPH B. L'EPISCOPO. Demonstrations of bone block operations for painful hips.
L. GASTON PAPAE. Acute epiphysitis of the hip joint.
CARL A. HETTESCHEIMER. Relief of lumbar backache by excision of the twelfth rib.

## NEUROSURGERY

*Jewish Hospital—Moderator:* LEO M. DAVIDOFF

E. JEFFERSON BROWDER and FLOYD BRAGDON. Physiological effects of drugs and hypertonic solutions in the normal and pathologic state.
RUSSELL MEYERS. Report on a series of operations for so-called "epilepsy."
RICHARD GRIMES. Review of 350 cases of fracture of the spine with and without spinal cord injury.
GUY LAUDIG. Report on 110 cases of subdural hematoma.
LEO M. DAVIDOFF. End-results in a series of brain tumor operations after ten to twelve years. Studies in electroencephalography and pneumo-encephalography.
ISADORE M. TARLOV. The brain and experimental lead poisoning. Tumors originating in the intracranial portions of the cranial nerves other than the acoustic nerve.
JOSEPH SIRIS. Studies on the blood brain barrier.
ANATOLE KOLODNY. Some infrequent spinal cord lesions; lantern slides.

## OTOLARYNGOLOGY

*Blood Stream Infections Complicating Ear Conditions —Brooklyn Eye and Ear Hospital—Moderator:* ROBERT L. MOORHEAD.

THOMAS B. WOOD. Variations in size and course of the lateral sinus.
CHARLES WEETH. Diagnosis.
MATTHEW G. GOLDEN. Results of sulfanilamide treatment.
PHILIP LEIBOWITZ. Management of infections.